THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—34TH YEAR. SYDNEY, SATURDAY, FEBRUARY 8, 1947. No. 6.

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THE PLACE OF MANIPULATIVE TREATMENT IN THERAPY.[1]

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IT is with some diffidence that I tackle the subject of manipulation and its place in therapeutics, knowing that I shall not be present to defend my thesis after my paper has been read. It is also difficult to discuss manipulation without being able to demonstrate points of technique as they arise. In spite of these handicaps, however, I welcome this opportunity of bringing this form of therapy to the notice of my colleagues in physical medicine in Australasia, for this branch of physiotherapy is one which is too frequently neglected and even more frequently dubbed by one's professional colleagues as "quackery".

It was in 1911 that my father's first book on treatment by mobilization was published. This mainly dealt with the treatment of fractures, but as the years have passed this book has run into five editions, the fourth and fifth editions being productions of this last war, and it now covers the whole field of physiotherapy so far as massage and manipulation are concerned. With wartime restrictions of paper this is a remarkable tribute to that work, but it is not for this reason that I draw your attention to it.

My reason for mentioning it is that the publishers tell us that the public demand in England, America, Canada, Australia and New Zealand indicated a considerable interest in the subject. It has been very gratifying to me to see the book on many shelves in Australasia, but I can find little evidence of its teaching being put into practice in New Zealand, and though I know my stay in Australia was so very short, I formed the opinion that there was small use made of manipulative treatment in physiotherapy. If I am wrong about this, I must apologize forthwith, but it is because of that impression that I suggested this subject as a suitable one for presentation to the first annual congress of the Australian Association of Physical Medicine.

After thirty years of fighting for the recognition of the rightful place of manipulation in therapy, it has at last been established that this is the treatment of choice for the painful stiff joint resulting from "adhesions"; but even today one not infrequently hears adverse criticism of this procedure and admissions of failure in its use, even in supposedly well-selected cases.

There remains, however, a large group of cases fer which we are convinced that manipulation is the correct treatment. It is for you to examine the claims that are made for manipulative treatment in these cases, and if you can be satisfied that there is a fundamental justification for them, then it is up to us to incorporate this method of treatment into accepted medical practice, thereby obviating the present apparent necessity for patients to seek advice from unqualified sources often at very considerable danger to their own well-being. That patients still seek relief of their symptoms from these sources is, to my mind, a slur on our profession in general and specialists in physical medicine in particular.

I have very little doubt that there is anyone present at this meeting who has not frequently heard friends or acquaintances extolling their pet osteopath or chiropractor who has cured them of pains of long standing when all the doctors have failed. There may even be one or two who have referred a patient to one of these practitioners. Certainly in England it is not uncommon for doctors to advise their patients to go to an osteopath, and just before I left Dr. Geoffrey Evans, in an address to the British Association of Physical Medicine, informed his audience that such was his invariable practice when he wanted a joint manipulated. I have already come across one patient in New Zealand to whom it was suggested by a very reputable doctor that he should seek help

[1] Read at the annual meeting of the Australian Association of Physical Medicine (British Medical Association) at Melbourne on September 5, 1946.

from a chiropractor as there was nothing further to be done for him in hospital.

There is no doubt that people are benefited and relieved of pain by manipulative manœuvres at joints where there is no evidence that any adhesions are present. It is unfortunate that members of our profession tend to pass off these stories of "miraculous cure" with the easy explanation that the patients are neurotic and their symptoms hysterical.

If manipulative treatment is a cure for hysteria, and a permanent one too, then it is strange that it has not come into vogue long before this as a universal treatment for a condition which most doctors can recognize quite easily and which, when they meet it in their practices, they all dread. Psychiatrists would all need to be proficient in the art, whereas in fact they have never seen any need to add manipulative treatment to their therapeutic resources. This alone should be sufficient to give the lie to this erroneous but common way of thinking.

We must therefore seek a more satisfactory and scientific explanation for these seemingly phenomenal manipulative cures. The crux of the matter was summarized by my father in a paper in 1934 and I cannot do better than to quote from it:

> Manipulative treatment means simply that some individual has performed on some other individual some movement, and that this movement has been administered with the avowed object of conveying a beneficial reaction. It is difficult to believe that any relief received by these means can have any other explanation save one of two. First, that mobility was impaired, that the impairment of mobility gave rise to symptoms and that these had disappeared as the direct result of restoration of mobility. The second possibility is that during the performance of the movement something was stretched or broken, and that the presence of this "something" intact had given rise to symptoms.

As I have remarked earlier, for this "something" we now substitute adhesions, and this is generally accepted as correct. It is the possibility that impairment of mobility gives rise to symptoms without the presence of limiting adhesions which I wish to discuss.

First, one must assume that a joint can have its function impaired without adhesions or joint disease being present. The evidence rests on the fact that instantaneous, lasting relief follows upon manipulation in this group of cases, whereas manipulation to restore mobility in a joint, limited in range of movement by adhesions, is almost invariably followed by some inflammatory reaction requiring sometimes weeks of careful after-treatment to complete the cure. When joint disease is present, manipulation produces even more disastrous results.

The medical profession has allowed the word subluxation to slip into its nomenclature. It is found in Gould's "Revised Medical Words" dictionary of 1928 and is defined as "partial dislocation". It is in this sense that it was used by Fairbank in 1925 when he was describing the condition of congenital partial dislocation of the hip joint. But it is not a word that one finds prominently in the index pages of textbooks. Yet it is quite common to hear the diagnosis of subluxation of a joint in which no partial dislocation can be demonstrated radiologically. In this sense the word is used to describe a condition in which a joint is immobilized or limited in movement in a position within the range of normal movement; it is just because the limit of mobility is within the normal range of movement, that it cannot be so demonstrated. Subluxation, used in this sense and founded on the principles of anatomy and the physiology of movement, is the term that I wish to justify, for only then can manipulative treatment as an orthodox therapy be completely accepted.

It is common knowledge that the ligaments which hold two bones together are not by any means always in the same degree of tension. Once again I will borrow from the paper I have already quoted:

> For example, in full pronation or supination of the forearm the inferior radio-ulnar ligaments are taut, whereas in the mid-position they are relatively lax. Thus it comes about that only in the mid-position can the radius be pulled down or pushed up in relation to the ulna in the long axis of the forearm. When so pulled down the two bones "lock" on one another and provide the clinical picture of the pulled elbow.

The "pushed elbow"—for example, after a fall on the outstretched hand—is far more common, though rarely described. Both afford admirable examples of conditions which yield to manipulation with a rapidity that often amounts to a so-called "miracle cure". Any forced movement of a joint while the ligaments are lax may cause a non-physiological movement to take place, and a "locking" of that joint is only what one might reasonably expect to follow.

Some practitioners will accept this explanation without question, but it is when they are asked to accept it concerning the joints of the spinal column that they become obstinate. Surely there is no fundamental difference between a joint of an extremity and the interlaminal vertebral joints, the costo-vertebral joints, the sacro-iliac joints and the occipito-atlantal joints. They all have the same anatomical make-up and they all move; in fact they are subject to more normal movement and abnormal stresses and strains than any joints of the extremities during a lifetime, for amongst other things, in every mile that a human being covers during his life these joints are apt to be exposed to unusual stresses at least a thousand times. If then we accept them as normal joints, it follows that they are liable in every way to the same damage as the joints of the extremities, and if we diagnose a traumatic synovitis of a knee or a traumatic arthritis in a shoulder joint following injury with resultant pain on movement and limitation of movement and function, then we must be prepared to accept the diagnosis of the same conditions in any single joint of the spine. Yet how seldom if ever do you hear the diagnosis of a traumatic arthritis of, say, the right lumbo-sacral joint, or a traumatic synovitis of the sixth left costo-vertebral joint? Subluxation of a sacro-iliac joint is a common diagnosis; there is no reason then why a subluxation of an interlaminal joint should not occur. Today many practitioners might recommend manipulative treatment to restore mobility to such a knee joint, shoulder joint or sacro-iliac joint, after the acute stage has settled down, and reasonably expect a cure. Few will yet confess that it is rational to prescribe the same treatment for similarly affected joints of the spine. Instead patients are referred for physiotherapy for backstrain, and they must count themselves lucky that Nature is such a wonderful healer that they get well in spite of and not because of what is done for them. How can treatment by physiotherapy be anything but empirical if physiotherapists give treatment for backstrain?

That manipulation often fails and sometimes does harm is irrelevant to the argument. The reason for lack of success is either faulty technique or mistaken diagnosis, and there is little difference between failure of manipulation from mistaken diagnosis and the lack of success attending on a laparotomy for a suspected perforation in a case of coronary thrombosis; but in this latter instance no doctor would be surprised that the treatment was a failure.

Whilst I am talking of manipulation of joints in which there are admittedly no limiting painful adhesions to break down, there would be a lack of completeness if no mention were made of visceral or referred pains or conditions of which patients claim to have been cured by manipulation, such as "sciatica" by manipulation of the lumbar part of the spine or sacro-iliac joints, "brachial neuritis" and, not infrequently, migraine and asthma, by manipulation of the cervical or upper thoracic segments of the spine, or more stupidly to us but realistically to them, of appendicitis, renal calculus, gall-bladder disease and even angina, which we know can have existed only in their minds because of faulty diagnosis.

Time will not allow a full explanation of all these miraculous tributes to manipulation, but three examples will suffice to indicate how careful one must be in diagnosis, and to show how in fact medical teaching already accepts the possibility that local pain may be

relieved by distant treatment. One of the symptoms of osteoarthritis of the hip joint is frequently said to be sciatica. This is inaccurate, for one very seldom if ever finds neurological evidence of sciatic neuritis in these cases. There may be wasting of the *quadriceps femoris* muscle and a diminished knee jerk, but never an absent ankle jerk or sensory skin changes. It cannot be disputed, however, that these patients suffer from pain of sciatic distribution and that relief from this pain can be achieved only by treatment of the hip joint, and manipulation may well be an essential part of this treatment. Conversely we are taught that pain in the knee is sometimes one of the first symptoms of tuberculosis of the hip joint. No amount of treatment to that knee will cure the pain or help the diseased hip, and obviously no active treatment of the hip will help the knee. Lastly it is commonplace that a symptom of gall-bladder disease may be pain in the back around the level of the seventh and eighth thoracic vertebræ. Manipulation will not cure cholecystitis; but if a gall-bladder can produce a localized segmental pain in the back, it is not much to ask that one should believe that a lesion or subluxation at a joint in the spine will produce a localized visceral symptom of pain. Certainly that is the only way in which to account for many of the magical cures of which we so often hear.

Why then does manipulative treatment fail? Why in so many professional circles has it still a bad name and does it fail to achieve its proper place in therapy that I would have you believe it should attain? There is only one answer to all these questions, and that is that there is no place for physical medicine in either the undergraduate or post-graduate curricula. Some time ago I was asked to submit a programme for a proposed post-graduate course in physical medicine. I did so, only to be told that it was thought that the subject was too specialized! Yet at one hospital with which I am acquainted, some eighty thousand odd treatments are given in the physiotherapy department in a year, and there is no specialist in physical medicine in charge of the department, such medical supervision as there is being delegated to junior physicians or an orthopædic surgeon when he has time to spare. The consequence is that general practitioners have not the remotest idea of the scope of physical medicine when adequately prescribed, and how can we hope for any improvement in the status of our speciality until we can give organized instruction in our subject?

The worst reputation held by any branch of physical medicine is that which manipulation enjoys, for above all therapeutic measures manipulation depends for its success on a technique which can be learned only by patient observation and study. But even before technique comes accurate diagnosis, and it is on the diagnostic side that our claim to consultant status must be founded. To support this I would like to give you an example of a fairly typical day's private work in London. Out of twelve to fourteen patients to be seen, four were new patients who had obviously been referred for an opinion as to their suitability for manipulative treatment. Two were suffering from backache, one from a stiff neck, and one complained of some foot trouble. They were all referred by doctors and all had come up from the country. One of those who complained of backache, a doctor's wife, was found to be suffering from a hypochromic microcytic anæmia, her hæmoglobin percentage being 40. She made a splendid recovery on iron medication and rest. The other patient I suggested was suffering from gall-bladder disease and she had no recurrence of symptoms following a cholecystectomy. The old gentleman with the stiff neck gave a history which made one suspect an infective arthritic condition, and on investigation was found to be suffering not only from sprue but also from a rare form of cystitis with prostatitis, and, of course, manipulation would have made him worse. The painful feet belonged to a man in whose case I was able to confirm a diagnosis of neurosyphilis and to put him on to the correct treatment which might be hoped to stop further deterioration of his condition. These cases speak for themselves in illustrating the dangers associated with the reference of patients direct to physiotherapists, to an unsupervised physiotherapy department, or worse still to an unqualified practitioner.

This is neither the time nor the place to discuss individual manipulations of joints, nor the technique appertaining to them. But this paper would be incomplete without a word on what manipulation hopes to achieve, the general rules of manipulation and its contraindications.

Manipulation in treatment is used to restore the normal range of movement in a joint functionally impaired. The two important words here are "normal" and "joint". We are all taught that at any joint the normal movements are flexion, extension, abduction, adduction and circumduction. These in fact are but half the normal movements at most joints, though they constitute the normal voluntary range. From a manipulative point of view there is an even more important group of movements which are normal to a joint, and these we call the normal involuntary movements. At a metacarpo-phalangeal joint there are four of these normal movements. These are long axis extension, antero-posterior gliding, side bending and rotation. No amount of manipulation will restore the normal voluntary movements if these normal involuntary movements are impaired, at least without the use of such force as would damage the joint structures to such a degree that the patient's last state would be worse than his first. A simple joint has been chosen for example, but the principle is common to every joint in the body.

The point relating to the word "joint" is obvious, but one which is all too often thoughtlessly overlooked. In manipulation we are treating one joint—or should be—not one limb or the whole spine. In other words every movement that we make must be a controlled normal movement at the joint we are treating. From this it follows that one aspect of the joint must be supported by a stabilizing hand, the other aspect must be moved on its opposing articular surface by a mobilizing hand and the movement must be a normal one. This is an inviolable rule if we are to avoid the pitfalls attendant upon uncontrolled manipulation. The only allowance that can be made is that the stabilizing agent need not necessarily be a hand.

The next rule is that the mobilizing movement must be a steady push or pull and not a forceful jerk. To achieve this the joint must be moved to its impaired limit and then the mobilizing effort is brought into play. More simply one "takes up the slack" and then pushes or pulls the joint through its first "dead point" when adhesions are present, or through its normal range if they are not. In dealing with adhesions it is of the utmost importance not to try to restore full mobility at one sitting, and it is a wise manipulator who stops after passing through this first "dead point". This may necessitate subsequent manipulations, but the more carefully you go, the more hope you may have of success.

It will be seen then that it is necessary not only to study the normal range of movement at any and every joint, but also to adopt a specific technique of manipulation of each joint.

The only real contraindication for manipulation of a joint is active disease at that joint, and by this is meant not only tuberculous, suppurative and specific arthritis, osteomyelitis, bone disease and neoplasms, but, more importantly even, those joint diseases which are almost certainly due to a low-grade infection not demonstrable by radiographic changes. Trauma is almost always a contributory ætiological factor in these cases. In such cases any focus of infection must be eradicated and the joint must be allowed to settle down. It is in these joints that adhesions always form, and to break them down while the joint is actively inflamed only courts disaster, as in the majority of cases injury of an inflamed joint encourages further inflammation, and almost every manipulation must cause some degree of trauma.

The most reliable guide whether it is the moment to manipulate is experience. If an infective process is present, there is likely to be a large amount of indefinite aching, and any pain which is caused by movement is sharp at first and then slowly subsides; the joint is more painful after rest; the pain is often of a throbbing nature. In the safe cases, which are those caused by simple trauma,

the pain will occur only as a result of movement as a sharp stab which is soon over and is lessened by rest. This point is of sufficient importance to be repeated. As a general rule it is unwise to manipulate a joint when there is a history of increased stiffness in the joint after a night's rest or after rest during the day. If rest does not give some relief to a joint, it is almost certain that manipulation will do that joint harm.

In conclusion, the man who talks of putting bones or vertebræ back into place is a danger, as is the man who practises manipulation empirically, forcefully and without technique, or whose only method of treatment is by manipulation. It is because such men exist, even alas in the ranks of our own profession, that manipulation in many quarters is held in disrepute. It is not until methods of physical treatment in general and manipulation in particular are adequately taught, understood and practised that patients may seek alleviation of symptoms at our hands with confidence that they will receive the skilled attention and treatment which they are accustomed to expect from our profession.

PHYSICAL MEDICINE AND ITS APPLICATION TO COMMON COMPLAINTS.[1]

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PHYSICAL MEDICINE is not so well known as it deserves to be. It is an essential part of general medicine, and in this country it must be a factor in our industrialization. Few medical courses include it, in spite of the part it has played and is still playing in the functional rehabilitation of the disabled in civil life as well as in the services. There is also an increasing demand by medical men for knowledge of a subject which has been neglected by those within the profession, and which has therefore been taken up, with handsome profit, by those without.

It is up to us as a profession, with the health of the community as our life work, to lead the way and impart the necessary knowledge, not only to the post-graduate student, which is now being done, but also to the undergraduate. It is also our duty to safeguard the public from exploitation for profit.

The elimination of all sense of the occult is possible only if the light of knowledge and experience is brought forward to expose the methods of the unqualified. For example, a patient comes along after having consulted those outside the medical profession with the following story: a painful knee has followed a blow with a piece of wood; the knee is swollen and obviously badly affected by old osteoarthritis; ten treatments by spinal adjustments naturally did not relieve the patient of anything but money. But on the other hand, we as a profession must be quick to learn and not deride all the methods of others. We must not, as we have done in the past, sometimes take little interest and discountenance what we hear because it is not customary or because it lacks scientific explanation.

Ten to fifteen years ago knowledge of the subject of physical medicine could be obtained by attending a post-graduate course in radiology, which included electrotherapy, and most of the large hospitals had good departments of physical therapy. At the present time we have in England a British Association of Physical Medicine and a Diploma of Physical Medicine, by means of which the medical man receives a good training in the physical principles and the technical aspects with experience in their clinical application. From America, where there has been for many years an active and progressive Association of Physical Medicine, much of our best work and knowledge has come. In England, before World War II, an Empire Rheumatism Council had been formed. With the coming of peace we hope that it will again take an active part in the study and treatment of rheumatism, one of the greatest causes of ill health in every country. With the formation of our Association of Physical Medicine (British Medical Association) in Australia it is hoped that a knowledge of the subject will be stimulated and made available to the medical profession.

Throughout the war the services have had a well-qualified and well-trained staff of physiotherapists, who have given efficient treatment at advanced units as well as at base hospitals. It was essential to rehabilitate all personnel as quickly as possible, and physiotherapists were used in every unit in the general post-operative treatment of wounds, injuries and surgical conditions. The amount of medical, surgical and orthopædic work was considerable, as it is in ordinary medical practice. Much of the plaster work was done by the physiotherapists. Considerable experience was gained in nerve lesions, and the plastic and facio-maxillary units had a full-time physiotherapist as part of the team.

In the medical wards a large amount of peripheral neuritis from deficiency disease and toxic conditions was a feature. Asthma, sinusitis, fibrositis, sciatica and rheumatism generally constituted a large amount of the work; exercises of a special nature for postural disorders, for neurological diseases, for psychiatric conditions *et cetera*, as well as general strengthening treatments, were given to many of the convalescent.

The whole problem of rehabilitation in the services has been and will continue to be for some time a field for the use of physical medicine. Rehabilitation has shown the necessity for physical therapy, and it must have its repercussions in the future. Coupled with this fact is the tendency towards socialized schemes in many countries. It is more than likely that the services of this specialized branch of medicine will play an important part in civil and industrial rehabilitation.[(1)]

As a whole it will be impossible to do justice to the subject. By eliminating theoretical and technical aspects and by selecting more the clinical side and some personal experiences, it may be possible to show what physical methods can be used to advantage in the treatment of disease such as is encountered in the usual run of medical practice.

The Objects of Physical Medicine.

What are the objects of physical medicine? They are: (i) to aid in the diagnosis and treatment of disease and dysfunction, (ii) to make familiar and to evaluate the various means and methods at our disposal, (iii) to determine which conditions can be alleviated by physical methods, (iv) to choose and direct the application of the appropriate method or methods.

The first object needs little comment. It is a pity that so much work goes to those who exploit a gullible public. Here it is that those outside the medical profession reap their harvest; but also members of the medical profession must be warned that the best results are obtained with any method of physical therapy only if the indications for its use are understood and some experience is gained in technical application.

The second, third and fourth objects need a little more consideration. There are many methods of treatment, some of which have been used for centuries. But the outlook is changing.

Forms of Physical Therapy.

The following are some forms of physical therapy.

The first type to be considered is thermotherapy, by either hyperthermy or hypothermy. Hyperthermy may be applied locally, by (i) hot compresses, poultices, water-bottles or sand, (ii) hot paraffin wax baths, (iii) infra-red rays and radiant heat, (iv) diathermy, or (v) short-wave therapy; it may also be applied generally by means of (i) hot-air baths, Turkish baths *et cetera*, (ii) hyper-

[1] Read at a meeting of the Victorian Branch of the British Medical Association on September 4, 1946.

pyrexia or fever therapy, (iii) air-conditioned cabinets. Hypothermy is applied by refrigeration anæsthesia or the ice-bag.

Electrotherapy is another form of physical therapy. It is given by (i) galvanism and ionization (direct current), (ii) faradism (electrodiagnosis), (iii) sinusoidal and other special currents, (iv) static electricity, (v) electrosurgery, (vi) diathermy and short-wave therapy.

Actinotherapy and heliotherapy are applied either generally or locally (Kromayer lamp).

Deep X-ray therapy is another form of physical therapy.

Spa therapy and hydrotherapy are given either (i) locally, by contrast baths, whirlpool baths *et cetera*, or (ii) generally, by means of baths, packs, sprays, pools, and such equipment as the Hubbard tank for underwater exercises.

Mechanotherapy is given in the forms of massage, manipulation, exercises (reconstructive and remedial), rest and relaxation and postural training, and by means of mechanical devices.

Occupational therapy, rehabilitation, physical fitness and physical education, and industrial medicine all come in the category of physical medicine.

The Application of Physical Medicine.

No more is it said: "Let the patient be electrified."

During the war of 1914-1918 electrical treatment was in its infancy. It was sometimes coupled with the X-ray department. A medical man who had been retired for twenty-eight years was put in charge of electrotherapy, because it was thought he could do no harm there, and treatments were given by a sergeant, whose only qualification was that in civil life he was a plumber.

Electrotherapy is an important and valuable part, but only a part, of physical medicine. The "colon laundries" have their place, as have the "hot boxes", Turkish baths, violet rays, short waves *et cetera*. You may be familiar with some of these methods. The particular value of each one, its specific action and purpose, require more than a superficial knowledge. It must be emphasized that apart from this specialized knowledge of the rationale of the method and how to apply it, a diagnosis, as accurate as possible, is first required, and some working knowledge of the pathological basis in each case.

The subject requires not only a specialized knowledge of some physical principles, but also a wide familiarity with medicine. Then and then only can the best work be done in physical medicine.

Take for example that common condition, pain in the arm. Often it is just labelled neuritis, without any idea of its causation. In the taking of the history and in the examination of the patient there are many points of importance—for example: the mode of onset of the pain, the type of pain, the site and distribution of the pain, the relation of pain to rest, the relation of pain to movement, any associated phenomena, any local tenderness, any limitation of movement of joints, any focal sepsis, the results of any special examination (for example, X-ray investigation), and any important points elicited in the general history and examination.

Many years ago the late Sir Richard Stawell gave an intensely interesting lecture on the mode of onset of pain. Relevant points were whether the onset was sudden or gradual and whether it followed any injury or illness. One must also determine what is the type of pain—acute, subacute or chronic, dull or throbbing—and its site and distribution. A pain in the hand may be referred from other structures or may be due to an irritation of the nerve roots in the neck or brachial plexus. Pain at the insertion of the deltoid commonly comes from the periarticular tissues of the shoulder joint.

It is important to know whether the pain is relieved by rest and what is its relation to movement. Dawbarn's sign is present if a catching pain is felt when the arm is abducted to shoulder level. It is increased by rotation of the humerus in abduction, and occurs in such conditions as supraspinatus tendonitis and calcification, and subacromial bursitis with or without calcification.

Any associated phenomena, such as "pins and needles", numbness and hyperæsthesia, should be noted. These suggest irritation of the nerves, whereas wasting may be due to disuse or it may indicate actual paralysis due to nerve injury or to true peripheral neuritis.

The presence of local tenderness often indicates an area of fibrositis, and pain may be referred from this area. Local tenderness may occur in muscle due to hyperæsthetic spots, and any pressure over bony ridges, over nerve endings in muscle, often elicits tenderness. Local tenderness must always be compared with the corresponding area on the other side. Pain accompanied by a red blush followed by the formation of vesicles is only too well known.

Any limitation of movements of the joints—for example, of the neck in the case of neuralgia or neuritis and of the shoulder joint when pain is largely felt in the upper part of the arm—should be noted. Probably the commonest of all conditions in this region which comes in for treatment by physical therapy is the presence of periarticular adhesions of the shoulder joint. The complaint is usually of pain in the region of the shoulder joint or at the insertion of the deltoid and down the arm to a more or less degree. Inability to lift the arm may be observed. There may be a history of injury, sometimes severe; the adhesions may follow a fracture or dislocation. Sometimes the injury may be due to indirect violence, such as a fall on the outstretched hand, or the injury may be so slight as not to be remembered by the patient. A pulled shoulder is not uncommon. A physiotherapist who is treating an elderly patient with an impacted fracture or a sprained wrist must always be warned to watch the shoulder. Some heat, massage and regular movements will prevent the development of adhesions.[2] Often so much attention is paid to the fracture with its splint, and to the pain and the position of the fragments, that the slight discomfort about the shoulder is neglected until some weeks later. By this time adhesions have developed and it is found that abduction or rotation of the shoulder joint is limited; this may mean a long period of treatment. This same condition of adhesions may follow an acute attack of neuritis, bursitis or fibrositis subsequent to a chill or some toxic condition.

When a patient with a stiff shoulder is first examined the question arises whether the joint should be manipulated and, if so, when.[3] Manipulation is practised in a very small percentage of such cases.

The following are some of the indications for manipulation.

1. Traumatic causation and absence of fracture.
2. Recent onset of the condition. Large muscular subjects give much trouble. The longer the condition has been present, the more dense and tough the adhesions. In manipulation considerable well-controlled force is required, the reaction is severe and subsequent active movements to maintain the range are difficult and in some cases impossible, even though the patient goes through torture. On the other hand, new adhesions are younger and less tough. It is sometimes advisable to break down adhesions in stages. There is always the temptation to do more whilst one is about it.
3. The absence of any toxic process. If a toxic process is present, pain is often acute—so acute that progress cannot be made with the usual physical therapy. Search diligently for a focus, eliminate it if present, and review the position in a few weeks. If pain is severe and no obvious focus is detected, a course of deep X-ray therapy is advisable. When the pain has settled down, satisfactory progress results.
4. Failure to respond to a fair trial of physical therapy.
5. The presence of pain only beyond the limit of range of movement.

The following are contraindications to manipulation: (i) satisfactory progress of the range of movement; (ii) the presence of a toxic focus (it nearly always means a severe reaction); (iii) the presence of calcification in the periarticular tissues and particularly in the capsule, as is seen in some old-standing cases; (iv) the presence of radiological evidence of even a slight degree of arthritis;

(v) failure of previous manipulations to improve the condition; (vi) the age and general condition of the patient; (vii) excessive pain.

Always have the range of movement measured each week under the same conditions and by the same masseuse, to avoid personal variations. The patient is much happier if he knows that his range is increasing, even though pain may still be considerable.

It is advisable to give the patient a trial of physical therapy for a few weeks before considering manipulation. Sometimes, because of some other conditions—for example, a weak heart, pulmonary tuberculosis or other disease—manipulation is out of the question. In other cases the patient simply refuses to have it. In these instances we have no choice.

It is usually found that gradually, if you have patience, the end result will be satisfactory. It may take six to twelve months, but treatments are not given frequently. It is advisable once a week to check up and give the patient some stretching, or to see him once a month to be sure that he is doing his exercises. This holds the patient's confidence. Too frequent treatment may defeat the object, especially in the case of women, who seem to become worn out with pain. Give them a holiday and then continue. Every patient must be treated individually. If manipulation must be carried out, do not be too enthusiastic. The resultant trauma is sometimes considerable and the reaction severe—even so severe as to make things worse. Here is a case in point.

A patient with a stiff shoulder had some physical therapy, and her shoulder was twice manipulated. After the second manipulation she had a completely "frozen shoulder". After some months of physical therapy she gradually obtained a painless shoulder with a full range of movements.

After manipulation immediate and intensive physical therapy is necessary. Place the patient in hospital for one week and have a masseuse attend twice a day to give short-wave therapy, massage and active movements. One-quarter of a grain of morphine is a help in enabling full movements to be obtained each day. Why is short-wave therapy necessary? The following history is illuminating.

A woman, aged fifty years, twisted her arm, and subsequently adhesions developed. For nine months she had tried hard to get relief by having massage, infra-red irradiation, passive and active movements and manipulation, both with and without anæsthesia. Pain was a constant feature and did not ease down until short-wave therapy was given. From then on this treatment, combined with massage and exercises, steadily improved the condition, and she was discharged from treatment, free of pain and with a full range of movements, in two and a half months.

The last restriction of movement, often affecting internal or external rotation, is sometimes slow to disappear, but persistence with exercises will overcome it. Some degree of pain is likely to be present so long as there is any restriction of movement, however slight.

Subacromial and Subdeltoid Bursitis.

Subacromial and subdeltoid bursitis is an unpleasant condition, but is fortunately not common. Pain is often acute and the condition has a fairly rapid onset after a period of mild pain. Usually a slight increase in the local temperature of the tissues is present, and often considerable swelling, with intense localized tenderness in the subdeltoid region just distal to the acromion process.

All active movements are impossible, but sometimes a full range can be obtained passively by the use of traction. It apparently pulls the inflamed tissues apart, and once one goes beyond the level of the shoulder, comparative ease is obtained. In subacute cases the main pain is experienced in abduction of the limb to the shoulder level, and particularly in rotation of the humerus in that position.

Treatment is by complete rest in bed in acute cases, but an abduction splint is not always helpful. Sometimes heat makes the condition worse, for an increase in the blood supply tends to increase the tension in the bursa. However, try heat treatment for twenty-four hours; if no relief is obtained, go straight on to deep X-ray therapy. This is usually rapidly successful. After one treatment the condition is bearable, and sometimes that is enough. If this is not available, an ice-bag will possibly reduce the tension and so relieve the pain. After the acute stage is over, heat should be applied and exercises given.

You may read of the use of low intensity short-wave therapy—so low that no appreciable heat is noticed. Most authorities do not agree with this form of treatment, and its rationale is hard to accept.

The subacute type of the condition usually responds to some form of heat. Movements are usually contraindicated, except just sufficient to prevent adhesions.

Calcification in the Supraspinatus or Supraspinatus Strain.

Calcification or strain in the supraspinatus may produce pain in the shoulder of variable intensity. Dawbarn's sign is present. There is usually no swelling unless the condition takes on an acute phase. Pain will be felt on certain movements, chiefly rotation in abduction. If an X-ray film reveals only slight density, then probably the condition will respond to short-wave therapy or deep X-ray therapy. The more chronic forms of the condition, in which a greater density of calcification is revealed by X-ray examination, often do not respond; but various treatments are possible in these cases.

1. Rest for six weeks in a plaster of Paris cast may be given. Probably adhesions will develop, but these are easily dealt with.

2. Rest in an abduction splint may be tried. This is but a compromise, and if rest is used, whether in a splint or in a plaster cast, the patient is out of action as far as earning his living is concerned. He will not take kindly to either method of treatment for a condition which is perhaps not very severe.

3. A needle may be inserted. It is even suggested that the area be washed out. It would be difficult to know whether the needle was in the right spot, and any good result is probably due to the "Novocain" and to the resultant reactionary hyperæmia which follows the insertion of the needle.

4. Surgical operation may be required, the area being exposed by reflecting the acromion process and deltoid. The exposure is excellent and once the calcified area is removed the results are good; but the operation is not so simple as may be imagined.

Tenosynovitis of the Biceps Tendon Sheath.

In tenosynovitis of the biceps tendon sheath there are localized pain with soft crepitus, pain on certain movements and sometimes limitation of movement. Trauma and infection are factors in the ætiology. Here again rest may be necessary, and heat treatment is sometimes unsatisfactory; but X-ray therapy and the injection of "Novocain" are often successful. The institution of massage or exercises usually irritates the condition, except when it is of the more chronic type.

Neuralgia.

Many cases of neuralgia are difficult to elucidate. Neuralgia is a term referring to an ache in some cutaneous area, but differing from neuritis. It may be a pain due to nerve irritation or referred from some source of irritation elsewhere—that is, referred pain. For example, fibrositis of the neck may start as acute stiffness of the neck with severe pain, which may later subside and extend down into the trapezius or upwards to the occiput, referred neuralgic pain being felt down the arm in the first case or upwards to the scalp in the second instance—the so-called occipital neuralgia with rheumatic headache.[4] Tender spots can be palpated in the muscles and relief will be obtained in various ways, as will be indicated later. The distribution of pain may be over the anterior or posterior thoracic nerves, affection of the latter not being at all uncommon; the irritation is in the region of the fifth and sixth cervical segments. Sometimes the pain may radiate down the arm to the hand and have various distributions, often radial. The irritation is frequently deep-seated from osteoarthritic changes interfering with

the normal foramina for the nerve roots, as is seen in an oblique X-ray film (Figure I).

Neuralgia may also come from deep-seated fibrositis involving the region of the brachial plexus.

The treatment in each case is a little different, for in the former the cause is a mechanical factor and treatment is by manipulation, whilst in the latter the cause is often a toxic focus and manipulation is contraindicated. Relief may be given by some form of physical therapy, but cure may not be obtained till the removal of the focus. In other cases the condition may follow some mild neck trick, a subclinical dislocation.

The method of manipulation with the Sayres head sling is most useful, immediate relief frequently being obtained with one treatment. Sometimes more than one treatment is needed. If the condition has been present for some time, all the muscles have been in spasm, and it takes a while for this to be overcome. The reason of preference for this method over that of manipulation under general anæsthesia is that it can be carried out simply in a minute or two, without the fuss, bother and upset of general anæsthesia and a period in hospital. It can be repeated as often as necessary, and the results are most gratifying. (Figures II, III, IV and V.)

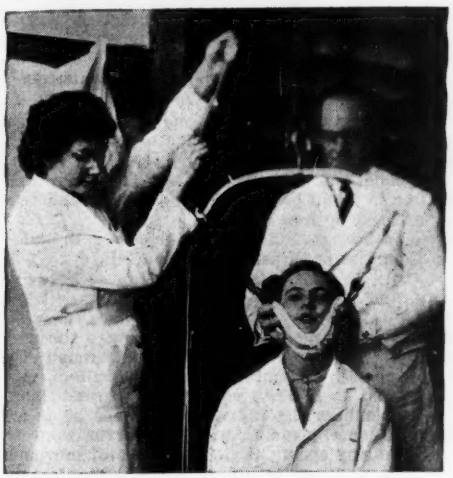

FIGURE II.

Success with infiltration of the cervical muscles with "Novocain" is uncertain; but treatment by manipulation, heat, massage and exercises is most satisfactory.

In other cases neuralgia is due to nerve root stretching or to pressure caused by faulty neck posture.[5] Acroparæsthesia may be a mechanically produced syndrome, and treatment by rest from arduous duties combined with active treatment by means of the Sayres sling and physical therapy is likely to be helpful.[6] Even the patient with a prolonged lateral process of the seventh cervical vertebra has been given ease by this method. Most of the neuralgias are treated in this way, the results are good, and it is rarely that manipulation under general anæsthesia is necessary or warranted.

Perineuritis.

Perineuritis is another entity which is an inflammatory condition of the fibrous tissue structures of the nerve sheaths and is often the result of chill or focal infection. It is really fibrositis, though it is sometimes known as interstitial neuritis.

Peripheral Neuritis or Neuronitis.

The true peripheral neuritis or neuronitis is a different condition with a different pathology. Acute brachial radiculitis is another term for a similar condition.[7] A sudden onset of pain, perhaps an elevation of the temperature, is followed by wasting and actual paralysis of muscles, more often in the shoulder girdle and in the upper part of the arm, less so in the forearm and hand. This may be associated with some virus. It was seen in many service men, among whom vitamin deficiency and tropical fevers were common. Recovery usually occurs, but it is slow.

Many patients on their arrival at the Heidelberg military hospital had received varying periods of infra-red irradiation, massage and remedial exercises, but showed more rapid improvement with short-wave therapy.

Again, there are cases with a toxic basis, such as alcoholic or diphtheritic neuritis. There are also the nerve lesions due to gunshot wounds or brachial plexus injuries. A study of these was made by Professor Sydney Sunderland in Melbourne.[8]

Here is the history of an interesting case:

Lifting a raft, a youth received a jolt by suddenly taking its weight. He went on swimming during the afternoon, but had severe pain that night followed by inability to use the arm. When he was examined some weeks after the injury, obvious wasting of the deltoid, the supraspinatus and the infraspinatus was present. Electrical reactions were those of degeneration. An abduction splint and physical therapy gave him complete cure in about four months.

FIGURE III.

Arthritis and Muscle Strain.

Arthritis and muscle strain need little comment.

Occupational or Habit Spasm.

Occupational or habit spasm is a condition in which the pain is localized to the *levator anguli scapulæ*, and is caused by continual elevation of the shoulder girdle due to habit or occupation. Cure is quickly obtained by explaining to the patient the method of production of the lesion and how to relax the muscle group.

Recent Work on Nerve Lesions.

Before we leave the subject of nerve lesions, recent work in this field is interesting. Since it has been shown that with physical therapy the degree of wasting and fibrosis can be minimized, a change of treatment has occurred. For years any condition of paralysis, such as poliomyelitis, Bell's palsy, neuritis and nerve lesions would not in any circumstances be treated with electrical stimulation. Such treatment has been blamed for contractions *et cetera*. The experimental work by Seddon at Oxford has shown that in muscles treated by electrical stimulation and massage, whilst the regeneration of the nerve was awaited, a quickened return to function, a better regeneration and less atrophy and fibrosis were observed.[9]

In any lower motor neurone lesion the electrical reactions are those of degeneration and are characteristic. The muscles are paralysed and soon waste. If the nerve damage is short-lived, we expect good recovery of muscle; if it is severe, then atrophy and fibrosis occur in the muscle, and this fibrosis is irreversible. It is a race between regeneration and fibrosis. So now stimulation is a popular form of treatment for all nerve lesions.

It is interesting to note that thirty years ago poliomyelitis was treated at Saint Bartholomew's Hospital with full-length sinusoidal baths, but fourteen years ago these were seldom used. It is possible that some similar form of treatment may again become fashionable.

Facial or Bell's Palsy.

Bell's palsy is so often due to œdema of the nerve in the facial canal caused by a focus of infection or toxic process such as influenza or dental or tonsillar sepsis, that almost invariably the use of short-wave therapy is indicated. Removal of the focus of infection has been known to cause a rapid change in otherwise intractable cases of several months' duration. The following cases are interesting.

A boy, aged seventeen years, had severe facial palsy which did not respond at all to four months' treatment by physical therapy. After removal of his tonsils an immediate return of function occurred, and he was discharged from treatment completely recovered within the space of a few weeks.

A woman, aged forty years, was examined four months after the onset of complete facial palsy. Practically no recovery had occurred. With the removal of a severe dental infection (pyorrhœa), she quickly regained function of the facial muscles and was discharged from treatment in about six weeks with a good result, only a slight contracture being left about the angle of the mouth.

The question of splinting arises. Splinting is permissible, but it does not alter or prevent the development of much of the atrophy and fibrosis which are responsible for the contracture. There is not sufficient evidence to be certain that stretching of paralysed muscles does harm. Fibrosis will always occur in a muscle if it is paralysed long enough and severely enough and is not treated.

Hundreds of facial palsies have been observed and treated; some have been splinted, some not; some have been stimulated, some not. The amount of contracture depends to a large extent on the degree of recovery of the nerve and on the length of time before any recovery takes place. It is a race between fibrosis and regeneration and fibrous tissues will contract. Splinting is employed in all severe cases, chiefly to prevent the eversion of the eyelid; but once any voluntary power appears, both splinting and stimulation are gradually reduced.

Spinal Conditions.

There are some conditions of the spine which are of interest.

A fairly common condition is that of pain in the dorsal region. It occurs more often in women than in men, perhaps owing to the fact that they are more often at the washtub, stooping over a sink or possibly just sitting. The patient is usually middle-aged. The chief symptom is often aching pain about the interscapular region, sometimes radiating round the thorax like intercostal neuralgia, sometimes boring through to the sternum. It is frequently worse when the patient is lying flat on the back or leaning against the back of a hard chair. Often an exaggeration of the normal kyphotic curve is present—that is, a definite fault in posture. Local tenderness may or may not be present, and the mobility is fairly good except for some slight diminution of rotation and hyperextension. An X-ray film may reveal little abnormality. Slight narrowing of the anterior portion of the bodies or the disks or slight lipping may be seen. As age advances and the condition progresses X-ray examination may reveal wedging of the vertebræ or pronounced osteoarthritic changes in perhaps one joint space. In many mild cases the condition cannot come into the category of either Scheurmann's disease or osteoarthritis. It is common to see it in orthopædic and physical therapy practices. The cause is probably prolonged faulty posture, either sitting or standing. Really the condition is a chronic strain, and when the patient is tired or not feeling fit, and as age advances, the droop occurs and strain results. This should normally be taken by the dorsal muscles; but if these are insufficient, the strain comes on the ligaments with perhaps referred pain or even dragging on the intercostal nerves. Dorsal neuralgia describes it well.

Treatment is fairly satisfactory. Firstly, manipulations are given to increase mobility; secondly, that mobility is maintained by exercises; thirdly, posture is improved by suitable reeducation; fourthly, short-wave therapy, which, because of its penetration, does seem to relieve pain, is employed; and finally, massage relieves any local spasm or tenderness.

Manipulation is really forcible rotation of the dorsal portion of the spine and springing of the vertebræ into hyperextension with the operator's knee in the middle of the patient's back.

While we are on the subject of posture there is much to be done in this field. Medical examination and physical education in the schools do a great deal of good. So also is it your duty in the home to see that the mother realizes the importance of good posture in her children. This is a worth-while part of preventive medicine.

In the streets and in one's usual practice many people are seen with neck forward, kyphotic back and everted feet, and even we ourselves adopt a "fireplace" attitude, with the consequent development of a waistline in the early forties. This is just carelessness.

To stand like that is partly made possible by the feet. It is difficult to stand badly, with the pelvis tilted and the abdomen relaxed, when the feet are straight. But with the "toes-turned-out" position of the feet it is easy and more comfortable to stand badly. Try it yourself, and remember the importance of foot posture.

Some months ago I heard on the air a British Broadcasting Commission broadcast, one of the "As I See It" series. C. B. Fry, of cricket fame, was speaking of skill at games, and he made the following points: "We British are congenital artists in our great games, considered of course as physical fine arts. We look up to the great men of sport: W. G. Grace, Trumper, Hobbs, Bradman *et cetera*." Some of you will remember famous footballers, golfers or tennis players. C. B. Fry's point was that nine times out of ten one remembers the individual because he is a stylist. "Nothing is more pleasing to the observer than to see a good style at any sport. Style and graceful movements are good to see and pretty to watch." Fry also used the following words:

Few people, even including some athletes, know how to stand well or walk well or how to hit well with any weapon, even if it is only a matter of knocking off the head of a thistle with a walking stick. There must be poise and bodily adjustment to achieve efficiency and natural grace cannot be achieved by mere conventional coaching. The fundamentals must be there. It happens that we are not without a good model to guide us in this matter. In all ways of physical skill and grace, the ancient Greeks attained the highest standard of fitness and skill and efficiency in the whole world—and what did they do? They trained their boys and girls from early childhood in fundamentals, how to place the feet and hands and how to stand. Their idea was that if the boy or girl was trained in physical requirements, they, or any or all of them, could go away and take up specialized pursuits with a certainty of success. So, after preliminary training in fundamentals, Greek girls and boys went away to find success in military skill and athletic prowess simply by going and doing it.

Ordinary conventional methods of training go so far, but may not tend to develop gracefulness, and may lack the fundamentals. This only goes to make the point about foot posture. To stand and walk correctly is a fundamental which must not be neglected.

Lumbar, Lumbo-Sacral and Sacro-Iliac Pain.

Lumbar, lumbo-sacral and sacro-iliac pain, with or without sciatica, are subjects about which much has been written and about which a greater knowledge has been

obtained. Whereas sciatica years ago was just a pain in the leg, it is now possible to be more accurate in discovering the cause or the mechanism of production. When this is established we are in a much better position to succeed in treatment.[10]

The use of "Novocain" has been most helpful in both diagnosis and treatment.[11] No more annoying condition comes for treatment than chronic backache. Consider all the causes of backache. We can eliminate certain causes, such as visceral referred pain or bony changes *et cetera*; but to find a localized tender spot in the sacrospinalis and then to relieve the patient of that particular pain by merely injecting five to ten millilitres of 0·5% "Novocain" solution gives information which helps considerably in

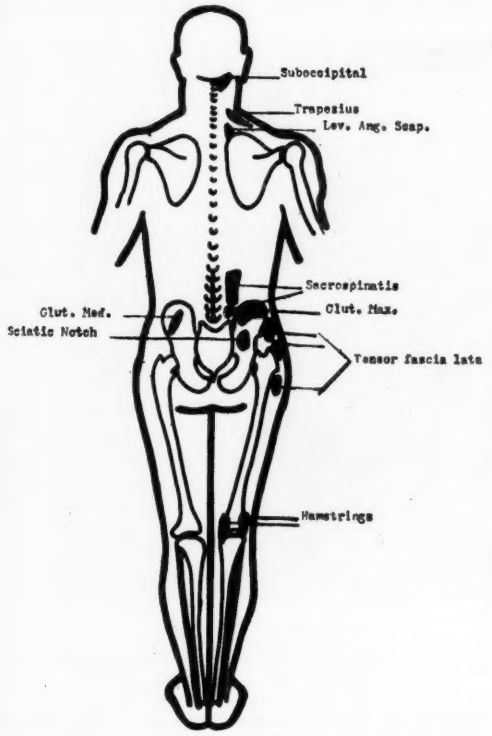

FIGURE VI.
Locations of frequent tender areas.

the confirming or making of an accurate diagnosis.[12] Furthermore, it is a good method of treatment. In many resistant cases the condition has responded dramatically to "Novocain" (Figure VI).

"Novocain", it is said, "acts by sympathetic nerve block with resultant vasodilation and improved circulation. Vasoconstriction may be the cause of the pain and the vasoconstriction is reflexly increased by the pain. 'The pain, so to speak, maintains itself.' Break this vicious circle, the pain ceases and circulation becomes normal." The same result may be obtained by several methods of physical therapy which are counterirritant. Infra-red rays, short-wave therapy, massage *et cetera*, all will relieve pain by vasodilatation, though the result may be slower.

Another condition commonly encountered is pain in the buttock, or gluteal fibrositis. This may follow an attack of acute lumbago. It is here that difficulty arises in diagnosis. It is certain that fibrositis may start in the lumbar region and spread to the gluteal muscles, from which referred sciatic pain may arise, or the inflammatory process may even go deeply into the region of the sciatic notch with the production of even more severe sciatica. But we also realize that an intervertebral disk lesion may start as lumbar pain, which later becomes more truly sciatic as the impingement extends to involve the nerve. A patient suffering from sciatica which can be relieved with the injection of 20 millilitres of 0·5% "Novocain" solution into the buttock probably has gluteal fibrositis—probably, because it is noticed in some intervertebral disk disorders that local tenderness is present in some muscles, due either to reflex pain and hyperæsthesia or to an actual protective spasm. The involved muscle is usually the sacro-spinalis or *gluteus medius*. Relief is obtained by injection of "Novocain" solution and this is confusing at times.

To return to the fibrositis; after "Novocain" injection the treatment is infra-red irradiation and deep massage followed by full movements and manipulation. This cuts considerably shorter the duration of this type of sciatica.

I have been called out many times to a patient suffering from sciatica, sometimes weeks, sometimes months in duration. I have gone armed with "Novocain" and a syringe, and an injection has been given. From then on the patient has been comfortable, has had a good night and has begun to recover.

Then there is the observation of relief stance. I notice that when impingement sciatica is present usually a relief stance is present also, so that the scoliosis in the lumbar region is convex to the affected side. In the presence of *gluteus medius* fibrositis and acute fibrositis of the buttock I have seen the opposite stance. I imagine this attitude tends to relieve the tension of the gluteal muscles and fascia with diminution of the pain. I have not seen it often, but I am inclined to think it is a helpful point in differential diagnosis of sciatica.

Sacro-Iliac Subluxation.

Does sacro-iliac subluxation occur? X-ray examination is no help. The patient complains of severe pain localized to one sacro-iliac joint, with referred pain down the posterior aspect of the thigh as far as the back of the knee, and no further. (If it does go further, I suspect an intervertebral disk lesion.) The mechanism of production is usually the lifting of a weight with the tension on the hamstrings, the tendency being to pull the pelvis down by the ischial protuberance. The sacrum is fixed with the spine. If one is looking at the left side of the patient, the torsion is clockwise, and if subluxation occurs, the movement is clockwise. To repair the displacement, put the affected parts in the opposite position. The patient lies supine; one lifts his leg and places it across the other leg so that it hangs over the edge of the couch; then with one hand on the patient's knee and the other hand on his shoulder (the same side, to stop the patient from rolling) one pushes; there is a click and the "miracle" is performed.

The other manipulation is more a rotary movement in a different plane. The patient lies on the opposite side. One hand is used to fix the patient's shoulder; with the elbow and forearm on the ilium, one pushes with considerable force; again, often a snap is heard and immediate improvement is noticed.

There are many tricks of manipulation by which those outside the medical profession make their name. They, of necessity, have developed these methods because they are not able to use anæsthesia. These tricks are of undoubted value, for many dramatic results can easily be obtained by them. A description of these methods is found in the literature.[14]

Before I leave this region let me say a word about our old *bête noire, morbus coxæ senilis*. Little can be done for it, but some patients are helped by physical therapy. Give them a trial for a time, and if gluteal tenderness is found, "Novocain" injection is helpful, especially in cases in which the pain is posterior rather than anterior.

Spondylitis Ankylosa.

Can we do anything for *spondylitis ankylosa*?[15] Yes, a great deal. It occurs far more frequently in men than in women—the ratio is about 50 to 1 in my experience.

It commences at an early age, about twenty to thirty years, and usually the first pain is in the sacral and sacro-iliac region. X-ray examination will reveal typical "fuzzy" joints and later some sclerosis. Later still comes the ossification of the ligaments of the spine (Figure VII).

Treatment may be by short-wave therapy, which sometimes gives relief; but reliance can be placed on deep X-ray therapy. This is only part of the treatment. If the spine is to ankylose, let it be straight, so that the patient can stand erect. Postural training and exercises are essential. Prevent flexion deformity at all costs. A low pillow and if necessary a brace are required. Breathing exercises are necessary to maintain the mobility of the thoracic cage. Attention should be paid to diet and tonics, and perhaps gold therapy may be indicated. The condition in many cases tends to become quiescent, but the acute cases are quite a problem.

Osteoarthritis of the Knee.

Let me say a few words about the osteoarthritic knee. Patients who have a bad mechanical factor due to "knock knees", with or without loss of cartilage on one side only, are not easy to help; but usually the combination of short-wave therapy, massage and non-weight-bearing exercises will give relief. Patients have been examined years after their treatment has ceased who have little or no pain, and yet it is known that the X-ray films revealed considerable osteoarthritis. Much of the pain comes from the peri-articular tissues.

A recent article reported the cure of periarticular fibrositis by injection of the hamstrings with "Novocain". This led me to try forcible kneading of the hamstrings as a means of relieving pain and stiffness of the knee joint. It is a painful procedure, but it takes only a minute and relief is rapid though not always lasting.

Sinusitis and Rheumatoid Arthritis.

It would not be possible to cover all conditions for which physical therapy is helpful and to open up such subjects as sinusitis and rheumatoid arthritis would be too much; but I should like to say that the allergic side of sinusitis and nasal allergy to foreign protein can be relieved in a great number of cases by two simple procedures, each requiring about three visits. These are local ultra-violet therapy to the nose with the Kromayer lamp, and zinc ionization of the nasal mucosa. About 70% to 80% of nasal allergy can be relieved in this way.[16]

Let me say a word about rheumatoid arthritis and its management.[17] Gold or physical therapy and routine attention to septic foci and general health are points in treatment which are well known; but a warning should be given about gold. It is better to delay the use of gold in the presence of inactivity of the disease and in the presence of focal sepsis. The blood sedimentation rate can be used as an index. It is not uncommon to find that a patient has been given gold for pain due to osteoarthritis and simple fibrositis. Gold has a toxic action and needs to be used in selected cases only and not given as a cure-all for pain. Doses should be kept to about 20 milligrammes.[18]

The whole subject of rheumatism needs research. We need to know what is the true pathological picture of fibrositis and what is the process that causes the swollen joints of rheumatoid arthritis—whether it is an allergic response to infection.

Let me refer once more to the septic focus. Many of you have seen good results from the removal of a tooth; many of you have seen no result from the removal of all teeth; but why? Often there is more than one focus, and unless you look you will not see. Firstly, consider pyorrhœa. This is a great menace and yet it is easy to detect: the red line of inflammation, the X-ray evidence, the hæmorrhage on squeezing of the spongy gums, the odour of the breath *et cetera* (Figure VIII). Before removal of the teeth see that they are scaled. The giving of sulphonamides or penicillin may prevent complications, and these drugs are now widely used.

The tonsils may be unhealthy. Obviously diseased tonsils present no difficulty, but buried tonsils are difficult. A history of repeated mild sore throat and the appearance of redness of the pillars or retention cysts probably indicate sepsis.

The cervix is a most important focus, for it is not always looked for. One must look for it. Even the so-called erosion, which is a different type of epithelium, can act as a focus. The condition may even occur in virgins.[19] The gynæcologist sees the erosion so often without symptoms of rheumatism that it may be disregarded; but a patient with rheumatism and an erosion must be regarded as a possible "winner". The cure is simple with cautery or ionization.

Rheumatism in men is frequently due to prostatitis. Examination of a smear will soon show the degree of inflammation. Many excellent results have been obtained by regular treatments once or twice a week with diathermy

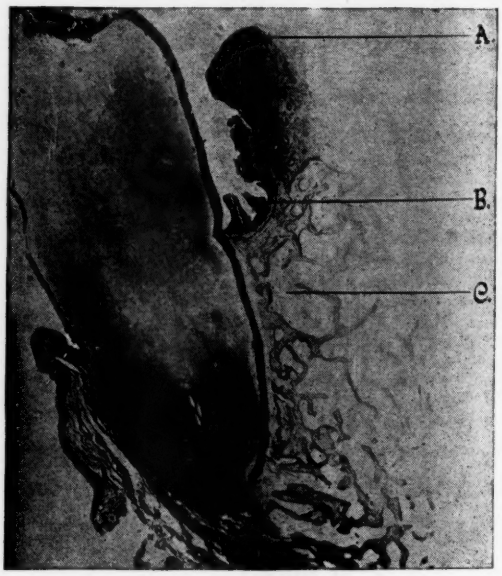

FIGURE VIII.
Photomicrograph of a pyorrhœal pocket. A = external epithelium. B = granulation tissue. C = alveolus. (By permission of Dr. J. Monahan Lewis.)

to the prostate and prostatic massage. This infection is often blood-borne and may be secondary to a more obvious infection, such as that of teeth and tonsils, which may have already been removed.

Ultra-Violet Light.

There are still some things which have not been mentioned—for example, ultra-violet light. This in Victoria has a definite value in physical medicine. Without going into details about physical principles, let me just say a few words about its use.

During the war in England, where everyone worked for long hours, with little time or opportunity for sport or leisure, where in the services men were overworked with night flying or in submarines *et cetera*, where men, women and children became debilitated from constant anxiety and poor living conditions, it was found that regular doses of ultra-violet light were beneficial.

In the modern community at peace there should be no debilitation any more than there should be rickets, for which ultra-violet light is so valuable. Poorly developed children suffering from broncho-sinusitis, asthma, pink disease and general debility respond satisfactorily to the addition of ultra-violet light to their general medical treatment. Particularly children suffering from sinusitis do well with all the routine methods of treatment—that is, short-wave therapy, general or local ultra-violet irradiation,

special and general exercises. I have had the opportunity of seeing sinusitis not respond to a course of short-wave therapy until the addition of ultra-violet light, when recovery was rapid.

Again, there are some skin diseases for which ultra-violet light is helpful, for example, psoriasis, alopecia, furunculosis, herpes, acne. For the elderly and in chronic diseases, such as rheumatoid arthritis and surgical tuberculosis, the value of ultra-violet light is undoubted.

Occupational Therapy.

Finally, let me say a word on occupational therapy, which is so linked with physical medicine that one basic educational foundation may even be required for both physical therapy and occupational therapy diplomas.

In the past much use was made of mechanical devices for producing passive and semi-active movements. Then came the period of active movements and exercises, and finally that of occupational therapy, which gives a mental stimulus as well as a definite plan for the rehabilitation of the unfit, instead of dull repetition of exercise without purpose.

Physical medicine is scientific and must go on in spite of criticism. It is up to us as leaders to see that all hocus-pocus is eliminated. In the planning of rehabilitation and preventive medicine we have a large part to play. In some countries the planning tends to comprise three main fields: physical medicine for diagnosis and treatment, occupational therapy and rehabilitation.

Electromyography.

Physical medicine has now become a distinct specialty in medicine, and we must look forward to and expect further developments. One of these will soon be with us—electromyography.[30] The cathode ray oscillograph has made this possible, for there is an action potential in the muscles which can be detected by the electromyograph, the amplitude of which increases with muscular effort. We can obtain a definite figure for each normal muscle and variations of the graph for the abnormal. Motor unit action potentials appear when muscles contract. They have 100 millivolts deflection and about 200 impulses per second. Fibrillation action potentials from denervated muscle are of shorter duration (1·2 milliseconds), lower amplitude and low frequency (two to ten per second). With reinnervation of the muscle they decrease before the return of motor unit action potentials, and functional recovery commences two weeks to six months after appearance of those potentials.

Electromyography is applied to the various groups of muscle and nerves, and we shall be able to apply it to the diagnosis and prognosis of muscular paralysis and nerve injury. It will provide a more delicate test of the condition of the motor nerve and it will be able to detect minimal degrees of lower motor neurone degeneration and the earliest stages of regeneration. It will not entirely replace the usual tests with the faradic and galvanic currents.

Conclusion.

In America there will be intensive research in physical medicine.[31] By the use of 100 million volts electron accelerators will be evolved giving radiations approaching cosmic rays.

Where we use six-metre short-wave therapy we shall have special electronic tubes giving high frequency power with a wave-length of only 50 centimetres. It will be possible to direct a beam of this energy towards any particular region of the body, and even to cross-fire it, as is now done with deep X-ray therapy. Electron microscopes will reveal the structure of protein and other extremely small and microscopic substances.

A close cooperation now exists in America between biological physicists and specialists in physical medicine, so that any new developments in biophysics will be available for research in medicine and for use in the practice of physical medicine.

Finally, a rehabilitation programme as carried out in military hospitals and ably explained to you recently by Dr. Douglas Galbraith, will give its proper place to each part of physical medicine in the reconditioning of the disabled, from the sick bed to the work bench.

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THE PITUITARY GLAND AND CARBOHYDRATE METABOLISM.

PART II: THE ANTERIOR LOBE.

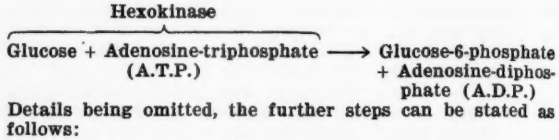
By A. B. CORKILL and J. F. NELSON,
From the Baker Medical Research Institute, Melbourne.

IN a previous publication from this institute, Nelson[1] described experiments concerning the anti-insulin action of anterior pituitary extracts. This investigation was concerned with the phenomenon that animals injected with anterior pituitary extract develop an insensitivity to the hypoglycæmic action of insulin. Studies were made on the isolated rat's diaphragm suspended in Ringer's solution in Warburg flasks according to the technique of Gemmill and Hamman.[2] By this method the diaphragm muscle is excised and immersed in cooled Ringer's solution (approximately 10° C.). It is then divided by cutting along the central ligament. The posterior portion is discarded and the two anterior portions are placed in separate Warburg vessels containing three millilitres of Ringer's solution; one section is used as a control and to the other insulin is added. The Warburg vessels are attached to the usual manometers, which are filled with 5% carbon dioxide and 95% oxygen before use. Under such conditions the strip in contact with insulin converts part of the glucose in the Ringer's solution to muscle glycogen. Strange to say, insulin does not produce increased oxygen consumption. Nelson[1] confirmed these findings, and in addition was able to show that in rats previously injected with anterior pituitary extract the action of insulin in promoting glycogen storage in the isolated muscle was inhibited. This was in agreement with the findings of Marks,[3] who studied the action of insulin in spinal eviscerated cats, injected some time before evisceration with anterior pituitary extract. In these it was observed that insulin was less effective in promoting the deposition of muscle glycogen.

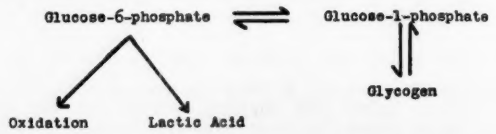
In certain quarters there is still a tendency to refer somewhat casually to the breakdown of glycogen to lactic acid or to the oxidation of glucose. It is not generally appreciated that an extremely complex set of reactions is involved in these transformations.

Harden and Young demonstrated the importance of phosphorylation (introduction of phosphorus into the molecule) of hexose in alcoholic fermentation, and in recent years it has been clearly shown that phosphorylation is essential to many metabolic processes, and in particular does this apply to carbohydrate metabolism. We now have the conception of energy rich phosphate bonds, in which large amounts of energy made available by metabolic processes accumulate. Creatine phosphate, which is of fundamental importance in muscular contraction, is a good example of a compound containing a high energy phosphate bond. During its enzymatic breakdown large amounts of heat are evolved. It is believed that by an elaborate series of transfers high energy phosphate bonds resulting from various metabolic processes are introduced into creatine.

The first step in the utilization of glucose depends on the enzyme hexokinase, which is responsible for the following reaction:



Details being omitted, the further steps can be stated as follows:



Cori[4] has recently shown that hexokinase activity can be inhibited in tissue extracts, either by injecting rats with anterior pituitary extract prior to the preparation of the extracts, or by adding anterior pituitary extract to the enzyme preparation. Further, it was found that such inhibition could be counteracted by insulin either *in vivo* or *in vitro*.

Although Cori observed that insulin released hexokinase from inhibition by anterior pituitary extract, he could not demonstrate that insulin increased hexokinase activity in tissue extracts. It must be remembered that our experiments are not strictly comparable with those of Cori, since he used tissue extracts, whereas we were concerned with the intact muscle. The experiments presented indicate that insulin promotes glycogen deposition by enhancing hexokinase activity, and that this influence is practically abolished by the previous administration of anterior pituitary extract.

Methods.

Gemmill's technique,[2] as previously modified by us,[1] was followed, the muscle strip being suspended in Ringer's solution containing 0·2% glucose. Rats weighing approximately 150 grammes were chosen, to ensure that the weight of each anterior portion of the diaphragm was in the vicinity of 0·1 gramme. Glycogen was estimated by the method of Good, Kramer and Somogyi.[5]

The anterior pituitary extract was prepared as follows:

The glands were removed and frozen within ten minutes after the animal's death. The anterior lobe was dissected out whilst still frozen and then ground for thirty minutes in ice-cold saline solution, two millilitres being used per gramme of gland. The suspension was centrifuged, the almost clear supernatant fluid was removed, and "Merthiolate" (1:10,000) was added.

One injection of 3·0 millilitres was given twenty-four hours before the experiment.

Results.

In further experiments, the results of which are shown in Table I, an attempt was made to obtain more precise information concerning the relationship between the glucose that disappeared and the glycogen that was deposited. From these data the following facts emerge:

1. The initial glycogen values from muscle of animals treated with anterior pituitary extract are lower than normal.

2. The glucose loss *per centum* from the Ringer's solution without insulin tested by strips from normal rats was 0·47. The mean of 62 determinations of oxygen consumption from previous experiments, which were strictly comparable to those now being reported, was 310 millilitres of oxygen at normal temperature and pressure per 100 grammes of tissue during a period of three hours. On the assumption that the respiratory quotient is 1, and from our own and Gemmill's findings, this seems a reasonable assumption; the oxygen consumption corresponds to the oxidation of 0·42 gramme of glucose. Hence 90% of the glucose that disappeared can be accounted for by oxidation.

3. With insulin, the extra glucose that disappears in strips from normal rats can be fully accounted for by deposition as muscle glycogen.

ILLUSTRATIONS TO THE ARTICLE BY DR. FRANK MAY.

FIGURE I.
Oblique X-ray film of cervical vertebræ, showing osteophytic lipping encroaching on foramina.

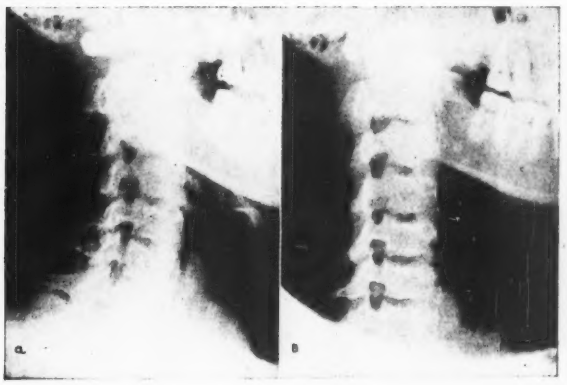


FIGURE IV.
Lateral views of the cervical vertebræ; (*a*) before traction was applied; (*b*) during the application of a traction of 90 pounds (41 kilograms); the normal cervical curve has been straightened, with resultant separation of vertebræ. (After McFarland and Krusen; from the White Memorial Hospital, Los Angeles.)

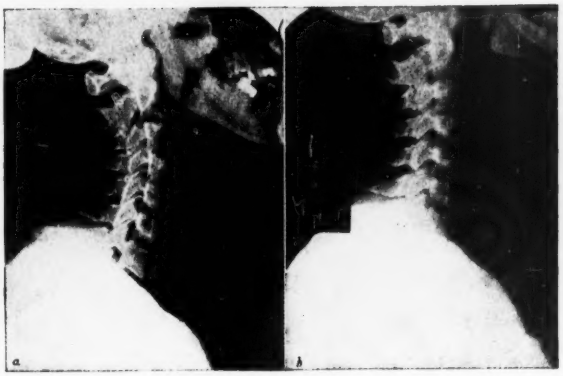


FIGURE V.
Three-quarter view of cervical portion of spinal column to demonstrate vertebral foramina; (*a*) before traction was applied; (*b*) during application of 85 pounds (38 kilograms) of traction; the cervical curve has been straightened, and the size of the foramina at the level of the fifth to the seventh vertebræ has been increased. (After McFarland and Krusen; from the White Memorial Hospital, Los Angeles.)

FIGURE VII.
Spondylitis ankylosa, showing sacro-iliac arthritis and ossification of ligaments.

ILLUSTRATIONS TO THE ARTICLE BY DR. C. Y. BLAND, DR. G. C. HUGHES AND DR. J. A. R. MURPHY.

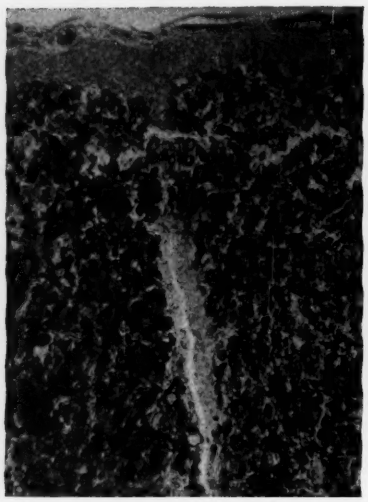


FIGURE I.

Dorso-lateral region of the spinal cord at the cervical level. Marchi stain. (× 100.)

FIGURE II.

Portion of Figure I. Shows myelin rings stained black with Marchi's stain and the absence of granular deposit. Areas most affected are seen between ground bundles and periphery. (× 200.)

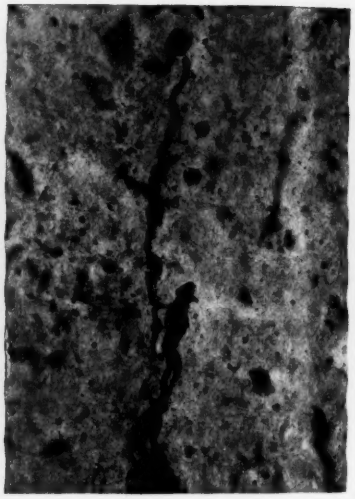


FIGURE III.

Lumbar area. Collaterals or very fine intercommunicating myelinated fibres in the grey matter show swelling of the myelin with beading and kinking of the fibres, also stained black with Marchi's stain. (× 200.)

ILLUSTRATIONS TO THE ARTICLE BY DR. M. L. EDWARDS.

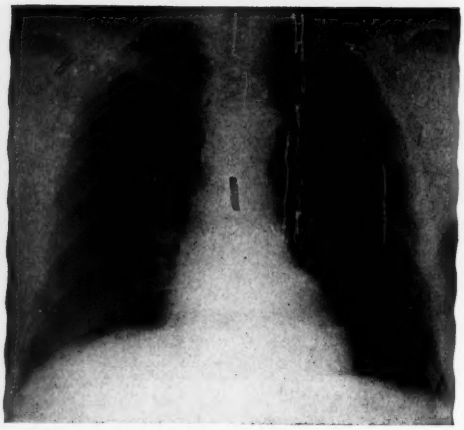


FIGURE I.

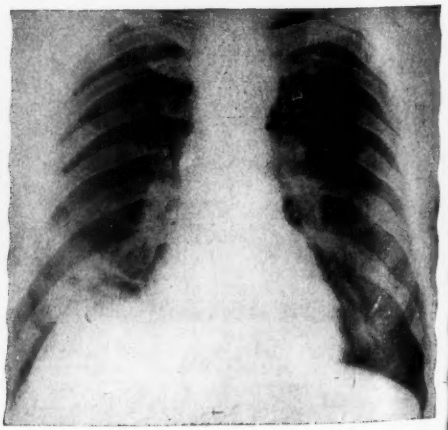


FIGURE II.

The amount of glucose that disappeared in the absence of insulin from strips prepared from rats treated with anterior pituitary extract is of the same order as that observed with normal animals. In the former group the value for extra glucose disappearing under the action of insulin is within the experimental error and is therefore negligible.

Discussion.

In the intact isolated muscle as exemplified by the rat's diaphragm, the action of insulin in promoting glycogen deposition can be explained on the basis that this hormone increases hexokinase activity and that this effect is abolished by anterior pituitary extract.

Without the results shown in Table I, it might have been argued that the effect of anterior pituitary extract in inhibiting glycogen deposition was due to another factor. Thus phosphorylation of glucose might have proceeded in a normal fashion with the formation of hexose-6-phosphate; but instead of proceeding via glucose-1-phosphate to glycogen, the reaction was directed to lactic acid.

In such circumstances one would expect an increased glucose disappearance, together with increased lactic acid formation. As is shown in Table I, in the anterior pituitary extract groups there is no significant increase in the amount of glucose lost. Furthermore, in some preliminary experiments there was no evidence of increased lactic acid formation.

At the moment we are unable to offer a satisfactory explanation for the finding that insulin, although promoting glycogen formation in the isolated diaphragm muscle of the rat, does not increase the oxygen consumption. The well-recognized effects of insulin are (*a*) increased oxidation of glucose, (*b*) increased deposition of glycogen in muscle, (*c*) regulation of gluconeogenesis from amino acids and fatty acids. Cori considers that either (*a*) or (*b*) may predominate according to the experimental conditions. In the experiments of Best *et alii*[6] with the spinal eviscerated cat, which to all intents and purposes may be regarded as a muscle preparation with its own intact blood supply, the disappearance of glucose from the blood stream under the influence of insulin can be accounted for by its deposition as muscle glycogen and oxidation. In our experiments we were also dealing with muscle, although here the normal blood supply was not present; but still it is difficult to advance a theory that will fully cover the facts.

Acknowledgements.

Our acknowledgements are due to Dr. F. G. Morgan, *Director of the Commonwealth Serum Laboratories*, for supplies of insulin used in these experiments, and to Mr. A. Doutch for preparing statistical analyses of the results presented in this paper.

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TABLE I.

Number.	Glucose Loss. (Percentage.) (*a*) Without Insulin.	(*b*) With Insulin.	(*c*) Increase in Glucose Loss with Insulin. ((*b*)-(*a*).)	Glycogen Percentage. (*d*) Without Insulin.	(*e*) With Insulin.	(*f*) Glycogen Deposition with Insulin. ((*e*)-(*d*).)	(*g*) Increase in Glucose Loss Unaccounted for by Glycogen Increase. ((*f*)-(*c*).)
Normal Rats.							
1	0·44	0·62	0·18	0·27	0·42	0·15	−0·03
2	0·50	0·79	0·29	0·33	0·64	0·31	+0·04
3	0·49	0·65	0·16	0·28	0·43	0·15	−0·01
4	0·45	0·94	0·49	*0·33*	*0·86*	*0·53*	*0·04*
5	0·64	0·82	0·18	0·10	0·30	0·20	0·02
6	0·34	0·53	0·19	0·32	0·53	0·21	0·02
7	*0·47*	*0·80*	*0·33*	*0·25*	*0·63*	0·38	0·05
8	0·49	0·80	0·31	0·32	0·67	*0·35*	*0·04*
9	0·55	0·78	0·23	0·44	0·69	0·25	0·02
10	0·40	0·64	0·24	0·33	0·53	0·20	−0·04
11	0·45	0·77	0·32	*0·41*	*0·76*	*0·35*	*0·03*
12	0·41	0·63	0·22	0·34	0·55	0·21	−0·01
13	0·45	0·63	0·18	0·26	0·48	0·22	0·04
Mean	0·47	0·72	0·26	0·31	0·58	0·27	+0·016
Rats Treated with Anterior Pituitary Extract.							
1	0·31	0·42	0·11	0·15	0·24	0·09	−0·02
2	*0·39*	*0·46*	0·07	0·16	0·19	0·03	−0·04
3	0·52	*0·58*	*0·06*	*0·14*	*0·20*	*0·06*	*0·00*
4	0·31	0·39	0·07	0·10	0·18	0·08	0·01
5	0·35	0·37	0·02	0·20	0·24	0·04	0·02
6	*0·49*	*0·58*	*0·09*	*0·17*	*0·24*	0·07	−0·02
7	0·60	0·65	0·05	0·22	0·24	*0·02*	*−0·03*
8	0·63	0·68	0·05	0·19	0·19	0·00	−0·05
9	0·36	0·40	0·04	0·24	0·26	0·02	−0·02
10	*0·68*	*0·72*	*0·04*	*0·23*	*0·25*	*0·02*	*−0·02*
11	0·65	0·67	0·03	0·13	0·15	0·02	−0·01
12	0·47	0·55	0·08	0·10	0·20	0·10	+0·02
13	*0·41*	*0·44*	*0·03*	0·13	0·12	−0·01	−0·04
14	0·47	0·52	*0·05*	*0·12*	*0·13*	*0·01*	*−0·04*
15	0·45	0·54	0·09	0·12	0·22	0·10	0·01
16	0·53	0·54	0·01	0·23	0·22	−0·01	−0·02
17	*0·30*	*0·37*	*0·07*	*0·14*	0·22	0·08	0·01
18	0·59	0·60	0·01	0·23	*0·27*	*0·04*	*0·03*
19	0·47	0·57	0·10	0·23	0·33	0·10	0·00
20	0·48	0·52	0·04	0·34	0·32	−0·02	−0·06
Mean	*0·47*	*0·53*	*0·06*	0·18	0·22	+0·04	−0·015

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HETEROSPECIFIC PREGNANCY: I. THE CLINICAL IMPORTANCE OF THE Rh FACTOR.

By P. M. DE BURGH, RUTH A. SANGER and R. J. WALSH,[1]
Sydney.

UNTIL methods for the detection of the incomplete antibody were discovered, it was difficult to prove that the Rh factor was the ætiological agent in the disease of any particular infant, in any stillbirth or in any miscarriage. The association of an Rh-positive fœtus and an Rh-negative mother indicated that an incompatibility existed, but direct proof of iso-immunization against the Rh factor was obtained in a minority of cases. Two important questions are raised by the results of the more comprehensive examinations of serum that are now possible; the first is whether an absence of evidence of iso-immunization excludes the Rh factor in the ætiology, and the second is whether the finding of iso-immunization necessarily means that the Rh factor is involved. It was held that the answers to these questions could be obtained only by a correlation of clinical and serological findings.

This correlation was attempted in a series of 54 cases, and although the number is relatively small it is thought that the results are significant. The histories were obtained from the obstetric hospitals at the time when blood was forwarded for examination. The tests for evidence of iso-immunization were performed by methods described in an earlier paper.(1)

Results.

The histories of the various patients, together with the results of tests for iso-immunization against the Rh factor, are shown in Table I. All the mothers were Rh-negative.

Comment.

Some of the patients amongst those showing evidence of iso-immunization do not present typical histories, and in some instances it is possible that the fœtal abnormality was not produced by the effects of iso-immunization.

Cases in which Iso-Immunization was Present.

Case 8.—The only pregnancies terminated spontaneously at six, seven and five months respectively. Whilst the Rh factor cannot be eliminated as a cause of the premature termination of all the pregnancies, it is possible that other factors were responsible in the first and even in the second instances.

Case 12.—The second child was a mongol. The third pregnancy resulted in the birth of a macerated fœtus on March 22, 1946. The mother's serum was examined on the same day. However, details of the pathological examination of this fœtus were not available.

Case 26.—The twins in this case were by a second husband. The unaffected infant was Rh-negative, whilst the other was Rh-positive.

Case 41.—Both infants died just before term and appeared comparatively normal. Autopsies were not performed.

Cases in which No Iso-Immunization was Present.

The cases in which iso-immunization was not found (cases 48 to 54) require more careful scrutiny.

Case 48.—Obstetric disproportion was found in this patient, and the first pregnancy terminated in a stillborn infant, presumably because of obstetric difficulties. The miscarriage in the second pregnancy was almost certainly unrelated to the Rh factor. The degree and duration of the jaundice in the third child are unknown; but no anæmia was found when the child was seven days old. The fourth child was normal.

Case 49.—The stillborn child from the second pregnancy died *in utero* at seven months. There was nothing to suggest that it suffered from hæmolytic disease.

Case 50.—The first child was delivered by the breech in 1939, and was not jaundiced or anæmic. The second child, now six years old, was born alive, but became jaundiced on the second day. The severity of the jaundice is not known, and no hæmatological examinations were performed. The third pregnancy terminated in a miscarriage at five and a half months, and the fourth in a miscarriage at six and a half months. The history of the patient is suggestive but not conclusive evidence of a blood group incompatibility.

Case 51.—The history in this case is suggestive of a blood group incompatibility between mother and children. The full history is as follows. (i) The patient had a miscarriage at the fourth month of pregnancy in October, 1921. (ii) A normal male child was born without difficulty in December, 1923. (iii) A female child was born in February, 1927; she became jaundiced on the second day, but the jaundice disappeared on the ninth day. On the eleventh day the child "collapsed", and on the seventeenth day she died in a convulsive seizure. (iv) A male child was born in December, 1929; jaundice was noted on the second day and persisted for approximately ten weeks; from the third to the sixth days almost continuous spasm was present. The child now has spastic tetraplegia. (v) A male child was born in June, 1935; jaundice was present at birth, and the child was transfused with the mother's blood daily for seven days; subsequently he received liver injections and liver and iron by mouth. There have been no permanent after-effects, and the child is normal and healthy. (vi) At the time when the serum was examined, the patient was two and a half months pregnant.

Case 52.—Both miscarriages occurred at five months, and the patient was four months pregnant at the time of examination. There is nothing to suggest that the miscarriages were due to a blood group incompatibility.

Case 53.—A diagnosis of hæmolytic disease of the newborn was made with regard to the child of the second pregnancy only because the blood contained a large number of nucleated red cells. There was no jaundice or anæmia. The diagnosis is therefore questionable.

Case 54.—Autopsy revealed that the last child died from inhalation of *liquor amnii*, and that there was no evidence of hæmolytic disease of the newborn.

Discussion.

The results recorded show that when evidence of iso-immunization against the Rh factor is obtained in an Rh-negative woman, the Rh-positive child causing the immunization was or will be affected by hæmolytic disease of the newborn. In this series there were two possible exceptions (Cases 8 and 12) to this general statement. As has already been stated, the history in Case 8 is not typical, and on the evidence available, one is not justified in concluding that the first or even the second miscarriage was the result of an Rh incompatibility. Nevertheless, the fact that the patient was immunized by the short-term pregnancies suggests an abnormal susceptibility to the antigen, and it is possible that at least the third miscarriage may have been due to this factor.

However, the finding of iso-immunization in a patient during pregnancy is not necessarily a poor prognostic sign for the unborn child. If the father is heterozygous the fœtus may be Rh-negative and unaffected, and the iso-immunization may be the result of a previous pregnancy or blood transfusion. This possibility must always be considered before the patient's confinement. Information is required on the changes which occur in the titre of the antibodies during pregnancy. A more precise knowledge of these changes may render them of prognostic value in any particular case.

The results are less enlightening on the question whether hæmolytic disease of the newborn may be due to Rh incompatibility in the absence of evidence of iso-

[1] From the New South Wales Red Cross Blood Transfusion Service.

TABLE I.

Pregnancies and Results.[1]

Case Number.	Number of Pregnancy.									Anti-Rh Agglutinin.	Incomplete Antibody.
	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	IX.		
1	N	N	N	HDD	P					+	−
2	N	HDD								+	+
3	N	HF								+	+
4	N	HF								+	−
5	N	N	HDS	P						+	−
6	N	N	M	HDS	P					+	−
7	N	HDD	M	P						+	−
8	M	M	M							+	+
9	N	N	N	HDS						+	−
			Twins								
10	N	HF	HF	P						+	+
11	N	M	M	N	SBHD	P				+	+
12	N	Mongol	SB							−	+
13	SBHD	HF								+	+
14	SB	HDS	HF							+	−
15	M	M	HDD	HDS	SB	P				+	+
16	N	HDS								+	+
17	N	N	N	N	N	HDS				+	+
18	M	N	SB	HDS						+	+
19	N	N	N	J	HDS					+	−
20	SB	J	SB	HDS						+	+
21	SB	HDS								−	+
22	D	SB	N	Twins	N	HDS	HDS			−	+
				D							
23	HDD	P								−	+
24	SBHD									−	+
25	N	HDD	SBHD	HDD	P					−	+
26	N	N	N	N	N	Twins				−	+
						1 N					
						1 HDS					
27	N	SB	M	SBHD						−	+
28	N	N	SB	P						−	+
29	N	N	N	N	N	M	HF			−	+
30	N	N	HDS	HDS						−	+
31	N	N	HDS							−	+
32	N	N	HDS							−	+
33	M	HDS								−	+
34	N	SB	HDD	SBHD	P					−	+
35	M	HDS								−	+
36	HF	P								−	+
37	N	HDD	SBHD	P						−	+
38	N	HF	HF	HF	HF	HF	HF	HF	P	−	+
39	SBHD	SBHD	HF	HF	P					−	+
40	N	SB	HDS							−	+
41	SB	SB								−	+
42	N	SB	N	HDS						−	+
43	SB	HDD	M	P						−	+
44	N	HDS	HDS							−	+
45	N	J	HDS	M	SB					−	+
46	M	N	D	M	HDS					−	+
47	M	HDS								−	+
48	SB	M	J	N						−	−
49	N	SB	J							−	−
50	SB	J	M	M						−	−
51	M	N	HDD	HDS	HDS	P				−	−
52	N	M	M	P						−	−
53	M	HDS								−	−
54	N	N	N	N	M	SB				−	−

[1] "N", normal infant; "M", miscarriage; "SB", stillborn—not definite hæmolytic disease; "SBHD", stillborn—hæmolytic disease; "HDD", death from hæmolytic disease; "HDS", hæmolytic disease with survival; "J", jaundice in neonatal period; "P", pregnant at time of examination; "HF", *hydrops fœtalis*; "D", death in neonatal period, not due to hæmolytic disease.

immunization revealed by present methods of testing. At first sight it would appear that the histories in Cases 50 and 51 could be explained only on the basis of such an incompatibility. However, it must be remembered that serum from the patient in Case 51 was examined eleven years after the previous pregnancy and when the present pregnancy had advanced only two and a half months. It is not unreasonable to suppose that any antibodies formed during the earlier pregnancy would have disappeared by this time, and that the sixth fœtus was not sufficiently advanced to produce antibodies. But the absence of immunization in Case 50 cannot be dismissed on these grounds. Whilst the history is suggestive of an Rh incompatibility, it is by no means typical. A more detailed account of the degree and duration of the jaundice and the result of hæmatological investigations in the second pregnancy would have been of invaluable assistance in this case, as in Case 48.

It would appear that as a general rule the finding of evidence of iso-immunization in a pregnant or parturient woman is closely correlated with the finding of some degree of hæmolytic disease of the newborn in the child; but this correlation is not absolute. The examination of the mother's serum before confinement is, therefore, of great prognostic value. The examination of her serum after confinement has diagnostic value, because hæmolytic disease of the newborn rarely if ever occurs in the absence of positive serological findings. The test described by Coombs, Mourant and Race[(2)] may be a more satisfactory early diagnostic procedure. This test indicates a specific union of antigen and antibody on the surface of the red cell and therefore the in-vivo iso-sensitization of the child's red cells.

Wiener[(3)] has assumed that the incomplete antibody is of smaller molecular size than is the anti-Rh agglutinin. He has suggested that the finding of an incomplete antibody is of graver prognostic significance than is the finding of the anti-Rh agglutinin, because the incomplete antibody would be more likely to pass across the placental barrier than the complete form. As far as can be ascertained, however, no attempt has been made to substantiate this latter suggestion by showing that the "index of placental permeability"[(4)] is less for the incomplete antibody than for the anti-Rh agglutinin. Furthermore, although the number of cases investigated in this series is not large, there is no evidence to support Wiener's suggestion. The theory of in-vitro dissociation of the altered complete antibody to the univalent form, proposed in an earlier paper,[(1)] is difficult to reconcile with Wiener's suggestion.

Summary.

The serological findings are correlated with the clinical histories of 54 mothers in whom Rh iso-immunization was suspected.

It is thought that hæmolytic disease in the infant is almost always associated with detectable evidence of iso-immunization when the disease is due to Rh incompatibility as the cause of fœtal or neonatal disorder.

Acknowledgements.

We wish to acknowledge the cooperation of those medical practitioners and hospitals who provided the clinical histories and blood samples used in the investigation.

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Reports of Cases.

A CASE OF THROMBOCYTOPENIC PURPURA AND GENERALIZED NEUROPATHY FOLLOWING SULPHADIAZINE THERAPY.

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THIS case is reported, firstly, because of the rarity of thrombocytopenia as a complication of sulphonamide therapy, and secondly, because the combination with neuropathy does not appear to have been previously recorded.

Clinical Record.

A.F., a male patient, aged sixty-one years, suffering from chronic pulmonary tuberculosis, was admitted to Waterfall Sanatorium on November 18, 1943. The clinical course was uneventful except for attacks of breathlessness, considered to be of cardiac origin. An electrocardiogram revealed pronounced right axis deviation, but no evidence of myocardial damage.

On April 8, 1945, he presented symptoms and signs suggesting left-sided pneumonia. Sulphadiazine therapy was begun on April 10, the dosage being two grammes for two doses and then one gramme four times a day until the seventh day of chemotherapy, when one gramme was given three times a day. Alkali and fluid intake were adequate. The temperature, from 102·8° F., returned to normal by the eighth day. On the tenth day a few petechiæ were observed on the trunk. On the eleventh day the petechiæ were more numerous, and the sulphadiazine therapy was suspended, a total of 38 grammes having been given. On the twelfth day the petechial areas had extended further, frank hæmaturia was present, and hæmorrhage occurred from the buccal mucosa, the tongue, the gums, the pharynx and the bowel. Examination of the blood revealed complete absence of platelets in the smear; the blood coagulation time (Lee and White) was fifteen minutes (normal limit, seven minutes); the bleeding time (Duke) was in excess of twelve minutes (normal limit, five minutes); the erythrocytes numbered 4,000,000 per cubic millimetre and the hæmoglobin value was 10·2 grammes *per centum*; the leucocytes numbered 10,500 per cubic millimetre, the percentage of neutrophile cells being 73, that of eosinophile cells 3·0, that of basophile cells 1·0, that of lymphocytes 16·0 and that of monocytes 7·0.

The patient was transferred to a metropolitan hospital, where on the fourteenth day of his illness a blood transfusion was given; the platelets then numbered 75,000 per cubic millimetre and the bleeding time (Duke) was sixteen minutes. Vitamin *K* was also given parenterally. By the twenty-eighth day the purpuric symptoms had completely subsided and the platelet count was then 226,000 per cubic millimetre. The patient was returned to the sanatorium in the sixth week, and was found to have evidence of generalized neuropathy. The clinical findings were as follows: some mental apathy and confusion were present, the cranial nerves were intact, the distal and proximal muscles of the extremities were weak and appeared equally wasted, the muscles of the trunk being normal; foot-drop and wrist-drop were pronounced, there was no dysdiadokokinesia and no tremor. Trophic disturbance with ulceration of the sacrum and heels was present; muscle coordination and the sense of spatial position were impaired; the sense of touch was impaired in the legs; pain, heat and cold sensations were impaired in the arms and legs, but not on the trunk; tenderness was present in the calves. The knee, ankle, triceps, biceps and wrist jerks were absent. Incontinence of urine was present for eight days during the sixth and seventh weeks. No abnormality was detected in the urine.

The patient was given a lengthy course of thiamin, nicotinic acid, dried brewer's yeast, liver extract and iron. By the sixteenth week the symptoms and signs of neuropathy had decreased. He was mentally normal, and he had good movement of the legs and arms in view of the muscle wasting, which was not noticeably relieved. The wrist-drop was the first to disappear; the foot-drop was much slower in recovery, and there was a residual drop of the right great toe. The trophic disturbances diminished rapidly and early, except for pressure effects on the hips and a slough on the left heel. Muscle coordination and spatial sense recovered early, together with the senses of pain and temperature. The tendon reflexes did not return to normal.

On September 2, 1945, the patient suddenly became very short of breath, lapsed into unconsciousness, and died within five minutes of acute cardiac failure.

Autopsy.

Autopsy was performed seven hours after death. The main findings were as follows.

The heart showed no evidence of distension or hypertrophy, the musculature being somewhat brown in colour and without evidence of infarction. The openings of the coronary vessels were of normal size, the vessels themselves being tortuous in their course and constricted at several points by localized areas of thickening. Typical atheromatous plaques were plentiful throughout the aorta.

In both lungs typical old fibrocaseous tuberculosis was present with extensive cavitation involving mainly the upper lobe of the left lung, which was shrunken and deformed. The lower lobe of the left lung contained patchy tuberculous consolidation of more recent origin.

The liver was firm, pale and mottled. The spleen was of normal size and consistency and contained a few irregular areas of dark red colour, though not of wedge shape. The kidneys were somewhat congested, but otherwise appeared normal. No gross changes were seen in the bone marrow of femur and sternum. The brain, meninges, spinal cord and peripheral nerves appeared normal.

Microscopic sections of various organs were examined by Dr. J. Tyrer. The left ventricle of the heart was affected by slight fatty infiltration and a slight increase in the subendothelial connective tissue. Atheroma was present in the left coronary artery; however, within the internal elastic lamina there was less evidence of degenerate material than is usual in atheroma. Intimal sclerotic changes were conspicuous. The right coronary artery was also affected by atheroma. An outstanding feature in the liver was the presence of cells of the leucocyte class, many of them situated in hepatic sinusoids. Their arrangement somewhat resembled that seen in hæmatopoietic areas in the liver of a fœtus. In some situations similar collections of cells of the leucocyte class

were to be seen in portal tracts where the connective tissue was increased in amount. Atrophic change in the parenchymatous tissue was rather conspicuous; it had an irregular distribution, and where it was most striking, the columns of epithelial cells were narrow and widely separated. In some of the epithelial cells of the liver fatty change was apparent. In the left kidney there was severe tubular damage; examination of the convoluted tubules showed not only intense cloudy swelling, but also in parts necrosis. It was difficult to decide how much of this was due to post-mortem change. In some of the glomerular tufts a slight increase in connective tissue was detected, while congestion was present throughout much of the kidney substance. In the spleen scattered hæmorrhagic areas and phagocytes containing altered blood pigment were found. A small group of tubercules, with giant cell systems, was present in one portion of the section. In sections of bone marrow from the sternum and femur, active hæmatopoiesis was apparent; but it was difficult to be certain of its exact nature.

A detailed microscopic examination of the nervous system was made by Dr. Oliver Latham. Sections were made from the Rolandic fissure, the optic thalamus, the brain stem, the *substantia nigra* and red nucleus, portion of the pons and medulla including the olive, the dentate nucleus of the cerebellum, and the cervical, thoracic and lumbar regions of the cord. The neurones in nearly all these areas appeared to be well preserved. Mallory's stain revealed a little glial increase in the bottom of the sulci, and a few neurones in the cord were affected by some axonal degeneration. However, such sensitive neurones as those in the dentate nuclei and olive appeared in excellent condition. Mallory's stain, which gives a good myelin effect, revealed no abnormality in the *cauda equina* or the anterior and posterior spinal nerves, save a thinning of the myelin fibres in one fasciculus of the peroneal nerve; if this was abnormal, it certainly was not recent. Staining by the Weigert-Pal and Robertson-Heller method revealed nothing amiss with the myelin anywhere, and other stains discovered no adventitial cells of any kind. By the Marchi method there was very little evidence of myelin change in any of the peripheral or spinal nerves—enough only to be classed as "slight". In pronounced contrast to this was the reaction in the posterior nerves and in the spinal cord itself after the cord had been entered. In both cervical and lumbar levels in the 36 sections mounted, extensive Marchi reactions were found in the myelinated fibres of the cord (Figures I and II); the condition apparently tended to affect all areas, but in the cervical level it rather spared the lateral and ventral pyramids and the area immediately internal to the periphery of the cord. The grey matter entirely escaped save for a number of very fine fibres apparently going through this region (Figure III) to arborize round the anterior horn cells, which themselves showed gross black staining of the pigment massed in them. A few of these cells were degenerated, atrophy and twisting of the processes being revealed, and some were "ghost" cells. The ground bundles seemed specially affected, and then there appeared a zone relatively free of change till another zone was reached which was deeply affected until it approached the periphery, which largely escaped. In the lumbar levels, however, the reverse was found. The peripheral zone, approximately one-third of the thickness of the cord, was the more deeply involved, especially in the dorsal tracts where the affection was intense. Wherever myelin fibres, thick or thin, could be seen in longitudinal section, the fibres were beaded and evidently degenerating. This was well seen in the grey matter. Some such fibres might be seen in the intracordal part of the fibres making up the anterior spinal nerves and also the dorsal. The staining was so like that obtained in Marchi preparations where definite tracts are involved that there is no reason to doubt the genuineness of the reaction, especially from the findings in the grey matter. No Marchi reaction was observed in the interstitial tissue, blood vessels or pia, but all fat globules took the stain well. There was no doubt that the patient had a moderate but definite fibrohyaline degeneration of his small vessels, especially in the cord; but the lumen of these vessels was always patent. Dr. Latham reported that he had seen far worse conditions obtain in arteriosclerosis.

It would seem, therefore, that the greatest damage to the patient occurred within the cord and was represented by a recent and wholesale myelin degeneration.

Comment.

Thrombocytopenic purpura is a rare but serious complication of sulphonamide therapy. Donaldson and Scarborough,[1] in a review of the literature up to June, 1945, found reports of only 22 cases. It has been observed as a complication of therapy with most of the commonly used members of the sulphonamide series, the dosage of the drug varying from three to forty-eight grammes, and the time of administration from two to eleven days. The above-mentioned authors have described an unusual case of thrombocytopenic purpura following the local application of 5% sulphathiazole ointment twice a day for two days. The mortality rate of the complication is high, and Rosenfeld and Feldman[2] recommend as a useful measure that when the regular blood smears from patients receiving sulphonamide therapy are being examined for possible granulocytopenia, a note as to the number of platelets present should be made, as this always decreases before symptoms become obvious.

Neuropathy, of varying type and degree, is on the other hand recognized as a common toxic effect following administration of sulphonamides. Psychotic manifestations, encephalopathy, encephalomyelitis, optic neuritis and peripheral neuropathy have each been frequently reported as a complication of this mode of therapy. It is possible that in the case described the neurological lesions were due to an intercurrent affection of the Guillain-Barré type; but the frequency of neurotoxic effects following sulphonamide therapy, delayed onset being not uncommon, strongly suggests that sulphadiazine was the causative agent.

Acknowledgements.

Our thanks are due to Dr. E. Sydney Morris, Director-General of Health in New South Wales, for permission to publish this paper; also to Dr. T. M. Greenaway, who was for a time medical attendant to the patient; to Dr. J. Tyrer and Dr. O. Latham, of the Department of Pathology, University of Sydney, for their kindness in carrying out microscopic examinations of tissue.

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AMŒBIC PERICARDITIS.

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IN recent comment on amœbic pericarditis[1] the rarity of the condition was stressed. For this reason the following case is considered worthy of record.

Clinical Record.

D.P.S., aged forty-four years, had seen service in Egypt, Syria and New Guinea. His previous history told of no significant illness. He had never suffered from dysentery, nor remembered being troubled by diarrhœa.

He was first admitted to a field hospital in a forward area of New Guinea on May 10, 1945. He complained that for the previous week he had suffered from subcostal pain on the right side, aggravated by deep breathing and passing to the neck. He had also a constant dull headache. Prior to this he had been well and active. On clinical examination no abnormality was found in the chest,

abdomen or circulatory system. Mild evening pyrexia from 99° to 100° F. occurred. No malarial parasites were found on repeated examinations of the blood. Ova of *Ankylostoma duodenale* were found in the fæces, and a routine vermifuge was accordingly given. His symptoms persisted, however, and on May 19 a pericardial friction rub was heard over the base of the heart and down to the fourth left intercostal space. The heart sounds were otherwise normal; no clinical evidence of cardiac enlargement or circulatory distress was noted. The blood pressure was 120 millimetres of mercury, systolic, and 78 millimetres, diastolic. At no time was there any significant fall in blood pressure. The leucocytes numbered 8,600 per cubic millimetre, and a differential leucocyte count gave normal findings. This pericardial friction, recorded by several observers, persisted until May 27, being described at first as "coarse and creaking in character", later as "harsh though faint". No electrocardiograph was then available in this forward area. On June 2 the persistent pain in the right hypochondrium had begun to radiate to the right shoulder, and tenderness was present in the right hypochondrium though the liver edge was not palpable. X-ray examination revealed clear lung fields and a regular diaphragmatic outline.

The patient was evacuated to the mainland by sea on June 9, eventually coming under my observation on June 26. He then stated that he was much more comfortable, after being at rest for six weeks, but some "catch" in the right hypochondrium persisted. No dyspepsia had occurred at any time. On examination, the patient's general condition was good; no clinical abnormality of lungs or circulatory system was found. The blood pressure was 115 millimetres of mercury, systolic, and 80 millimetres, diastolic. There was no abdominal tenderness, and the liver was not palpable, nor was its area of dulness to percussion increased. Jaundice was at no time observed. An electrocardiogram revealed a diphasic *T* wave in Lead II and some left axis deviation, but the findings were otherwise normal. Subsequent tracings were similar. A cholecystogram revealed a normally functioning gall-bladder, with no evidence of calculi. As the patient was now afebrile and pain appeared only slight, he was allowed moderate activity out of bed. Symptoms of diaphragmatic irritation, however, gradually increased. Finally, in an X-ray film of the chest on July 18 (Figure I) there appeared a rounded, localized elevation of the diaphragm on the right side just lateral to the cardiophrenic angle, which had not been present on previous examination. The leucocyte count was now 10,000 per cubic millimetre, 66% being neutrophile cells, 6% metamyelocytes, 23% lymphocytes, 4% monocytes and 1% eosinophile cells. Although no amœba or cysts had been found on repeated examination of the fæces, it was considered that the empirical administration of emetine was indicated. The intramuscular injection of emetine hydrochloride, one grain per day for ten doses, was begun on July 20. This was later followed by the routine army course of treatment with emetine bismuth iodide and "Carbarsone". On July 23 the patient was troubled for the first time by cough, with frothy, purulent, blood-stained sputum. On the following day the sputum was brownish, thick and copious, over ten ounces being produced in twenty-four hours. Microscopic examination of the sputum revealed only pus cells, epithelial cells, some red blood cells, debris and a few long chains of Gram-positive cocci. Examination of the chest at this time revealed patchy consolidation of the lower lobe of the right lung, with coarse pleural friction laterally and frequent rales. For a few days mild pyrexia was present, the temperature never rising above 100° F.; but the clinical appearance at no time suggested the toxæmia of primary pneumonia. Sulphadiazine was given by mouth, 36 grammes from July 23 to 28. By July 30 all symptoms had greatly diminished, cough was negligible and sputum consisted only of scanty mucus. On August 9 the patient was free from all symptoms and feeling well. The radiological report on his chest (Figure II) stated that the right side of the diaphragm was elevated in the middle of its leaf. At the base of the right lung there was an area of opacity in the lung field overlying this elevation with increased vascular markings running to the right hilum. The appearances were consistent with a rupture through the diaphragm leaving residual pneumonia and adherent pleura.

Further convalescence was uneventful. X-ray examination of the chest on October 3 revealed only slight residual pleural thickening. On October 5 he was discharged to duty on the mainland, feeling well.

Comment.

On clinical evidence there appears no doubt that this case was one of amœbic abscess of the liver, with spontaneous rupture through the diaphragm. This is supported by the X-ray evidence, the character of the sputum and its sudden copious expectoration, the absence throughout of the toxæmia which would necessarily ensue from a bacterial cause, and the satisfactory response to specific therapy.

The chief interest of the case lies in the persistence during the early stages of the illness of pericardial friction, and the fact that at the time this was the only clinical abnormality evident. It is suggested that this may reasonably be interpreted as evidence of a pericardial reaction to the underlying amœbic infestation of the liver.

Acknowledgement.

My thanks are due to Major-General S. R. Burston, Director-General of Medical Services, for permission to publish this report.

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(1) "Amœbic Pericarditis", THE MEDICAL JOURNAL OF AUSTRALIA, February 2, 1946, page 159.

Reviews.

THE DIAGNOSIS AND TREATMENT OF PEPTIC ULCER.

HELD AND GOLDBLOOM'S recent publication, "Peptic Ulcer: Its Diagnosis and Treatment", is disappointing.[1] Many recent aspects of treatment are discussed lightly or not at all. The bibliography, although large, contains a number of old and outmoded references.

Pathogenesis is discussed in the opening chapter, but, like others, the authors are unable to make any definite statements. They tend to regard the constitutional factor as very important.

The symptoms are well described and the importance of careful history taking is stressed as being of more value than the physical examination which so often gives negative results. Many physicians will not agree with the statement that "the same time interval elapses between the meal and the occurrence of pain regardless of whether the ulcer is situated in the pars media near the lesser curvature of the stomach, or in the pylorus or duodenum".

The chapter dealing with radiological diagnosis presents a number of clear plates of interest to the physician.

Many will disagree with the authors in the importance they attach to the role of recurrent appendicitis in causing symptoms resembling those of peptic ulcer. Even the fact that symptoms continue after appendicectomy, does not, according to the authors, rule out recurrent appendicitis as their cause.

They strongly recommend sodium bicarbonate as an antacid, though general opinion is against its use, while colloidal aluminium, now recognized as an ideal neutralizer of acidity, is disregarded. The contentious subject of smoking is avoided. "Larostidin" is mentioned as having been found beneficial, but the authors do not state that later results showed it to be valueless. Penetrating and perforated ulcers are discussed in a comprehensive manner.

The treatment of hæmatemesis is discussed, but the authors tend to be hesitant in the use of blood transfusions. The long period of bed rest (six weeks) is not in accord with most modern opinions, and its risks, such as embolism and the development of a neurosis, have to be seriously considered.

The authors replace the term "pyloric stenosis" by the words "gastric motor insufficiency"—a rather unnecessary addition of new terminology as the primary lesion is undoubtedly pyloric stenosis.

[1] "Peptic Ulcer: Its Diagnosis and Treatment", by I. W. Held, M.D., F.A.C.P., and A. Allen Goldbloom, M.D., F.A.C.P.; 1946. Springfield, Illinois: Charles C. Thomas. 9¼" × 6", pp. 394, with many illustrations. Price: $6.50.

The Medical Journal of Australia

SATURDAY, FEBRUARY 8, 1947.

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THE LUNACY ACT OF NEW SOUTH WALES.

AMENDMENTS have recently been made to the *Lunacy Act*, 1898-1945, of New South Wales, and careful scrutiny of the amended Act reveals anomalies that are fraught with danger to the community. The possibilities that may arise under the Act as amended are so grave that non-medical persons have drawn the attention of the Council of the New South Wales Branch of the British Medical Association to them. An additional clause (67A) has been added to the Act. Subclause 1 of this clause is as follows:

> In any case in which a person charged with an offence for which he has not been tried (hereinafter called "the patient") is detained in a hospital for the insane or in a hospital for the criminal insane the Attorney-General, if he is of the opinion that the question whether the patient is fit to plead if put upon his trial should be determined by a jury, may by order under his hand direct that the patient be removed from the hospital in which he is detained to some gaol appointed by the Attorney-General and specified in the order.

Subclauses 8, 9, 10, and 11 of this new clause 67A read as follows:

> (8) If the jury find that the patient is not fit to plead he shall be returned to the hospital from which he was removed.
> (9) If the jury find that the patient is fit to plead he shall be returned to the gaol.
> (10) If a bill is found against him he shall be placed upon his trial.
> (11) If a bill is not found against him a certificate shall be issued by the Attorney-General to the judges of the Supreme Court in accordance with the provision of section three hundred and fifty-eight of the Crimes Act, 1900, and any of the said judges may thereupon direct the gaoler in whose custody the patient may be to discharge him from custody.

It has been represented to the Council of the New South Wales Branch that the determination whether a person charged with an offence for which he had not been tried, and detained in a hospital for the insane or a hospital for the criminal insane was fit to plead if put upon his trial, was not one for a jury, but for some other body such as a panel of medical practitioners. It is quite possible that a jury might find a person fit to plead even though expert medical witnesses held a contrary opinion. The new subclauses also show that an insane person who had been found fit to plead and was found not guilty of the crime for which he was charged, might possibly obtain his release. Another point that has been raised is whether there is need for hospitals for the criminal insane. Whilst it is realized that there is need for the segregation of insane persons who may be dangerous, the question has been asked whether there is justification for the detention of these persons in institutions designated as hospitals for the criminal insane.

The Council of the New South Wales Branch considered this matter and referred it to the Section of Neurology, Psychiatry and Neuro-Surgery. The Section has expressed an opinion as follows. In the first place juries as at present chosen are not capable of making decisions as to fitness to plead; a special jury should be empanelled, but this should not consist of medical practitioners. In the second place when cases of fitness to plead are being tried the judge should have to assist him a psychiatric assessor or assessors. Thirdly, when a person who is insane is found not guilty of a crime he should still be considered insane in terms of the *Lunacy Act* and should remain under the jurisdiction of the Inspector-General of the Insane until he is deemed fit to be discharged. Fourthly, criminal mental hospitals are essential for the care and detention of insane criminals convicted of serious crimes. In the last place the Section thought that when a person charged with an offence and remanded was suspected of being of unsound mind, his fitness to plead should be determined before an order was issued by the Colonial Secretary under Section 67 for admission to a mental hospital or a criminal mental hospital. In reaching their conclusions the members of the Section availed themselves of special knowledge possessed by some of their number of cases which have recently been before the courts.

The position does not require to be argued at any length. The *Lunacy Act* has been amended and as it now stands it is possible that an insane person found fit to plead may, on being found not guilty of a crime with which he is charged, gain his release. The anomaly should be corrected at once so that, if release does take place, it takes place from the care of the Inspector-General of the Insane and not from a court that has been trying a criminal charge.

"THE SPIRIT IN WHICH WE WORK."

THESE are days when it is of the greatest importance for medical men and women to take stock of themselves and of their motives—that they may be quite certain that their motives are worthy of the highest tradition of medicine and that they may in the eyes of the public be examples of the type of living that they claim to represent. Nowadays we hear a great deal about the doctor-patient relationship and the urgent need for its preservation. We have to see to it that the relationship as created by each of us is worth preserving. Sir Alfred Webb-Johnson, the President of the Royal College of Surgeons of England, gave an address at Liverpool Cathedral, England, on "The Spirit in which we Work". A short account of this address is given in the *British Medical Journal* of October 26, 1946, and also in *The Lancet* of the same date. These abstracts which differ in certain respects should be read by all to whom

they are available. The message is chiefly for the members of the profession, but the speaker had something for the general public. He said that there was anxiety, now that the State was about to take a real part in British medicine, lest the great administrative machine to be set up might dim the high ideals of the medical profession or mar the close relationship that should exist between doctor and patient. The doctor's service was essentially personal and had to be given, not when he chose to give it, but daily at the need of others. The well-equipped physician had to possess the qualities of the artist, the man of science and the humanist, but he had to exercise them only in so far as they subserved the recovery of the individual patient. He had to feel directly responsible to the patient, not for him to someone else. The speaker went on to say that it was a hard doctrine, but none the less true, that this essential function of the doctor might involve the forgoing of exactly scientific diagnosis, of the perfecting of an operation, or even of the interests of society at large. The doctor himself has to realize that he will be able to fulfil his essential function only if he sees himself as he really is in relation to the Cosmos. Without this understanding, this vision, he may do certain things for those with whom he comes into contact, but he will not bring to those around him any knowledge, or even any glimmering, of the wider life of which he and they should be conscious parts. Man lives not for today or tomorrow or even for a lifetime; he lives, in some fashion that he does not understand, for eternity. Sir Alfred Webb-Johnson had thoughts something like these, for he said that the laboratory could satisfy many of our needs, but not those of the spirit. Therefore he added:

> Let knowledge grow from more to more,
> But more of reverence in us dwell.

He said that belief was the healthy act of a man's mind; and he asked whether every true man did not feel that he was himself made higher by doing reverence to what was really above him. "In order to realize the moral responsibilities of science we must look to something higher, and something beyond our own little lives."

Current Comment.

FEBRILE HEADACHE.

HEADACHE is so frequent a symptom in febrile illnesses that it has aroused little curiosity. Its type and distribution have been described in specific infections, and its peculiar insistence and frequency in certain illnesses, such, for example, as typhus, have also been often noted. Work has also been done on the possible association of headache with changes in pressure and composition of the cerebro-spinal fluid, though this has yielded little information except in those infections in which the meninges are involved in an inflammatory process. More interesting and important are the observations made by several workers on the amplitude of the intracranial and extracranial blood vessels. This work has been carried out both in the clinical and in the experimental field. R. B. Scott and R. P. Warin have published an interesting series of clinical observations on the nature and on the possible causes of headache and eyeache studied in military patients in the Middle East.[1] Their records are compiled from investigation of 100 soldiers. Of these, 50 had sandfly fever, 28 benign tertian malaria, 11 a brief fever of undetermined nature, and the rest various other common infections. So far as their studies go, these workers have formed the opinion that the mechanism of headache does not vary with the type of the infection. They refer to the work of Pickering on the influence of histamine in producing headache, and of Sunderland and Wolff on the similar effect of foreign protein introduced into the circulation. In the latter work dilatation of arteries has actually been seen through a window in an animal's skull. There is also evidence of the bearing of arterial blood pressure on headache, such as the observation that the pain is increased by any act which suddenly raises the blood pressure.

Scott and Warin charted the areas where pain was felt and found that three were sharply defined: bitemporal and vertical, occipital, and infranuchal. The choice of these fields varied with different infections, and in most cases pain was equal on each side. Headache and pain in the eyes were not necessarily related. Headache was found to correspond closely with the duration and degree of fever, and with prominence and vigorous pulsation of the temporal arteries and of the retinal arteries, and also with capillary pulsation in the skin. No constant changes were found in blood pressure readings. Other observations bore out the arterial origin of the headache. Obliteration of one common carotid artery relieved the pain on the same side of the head; but the headache returned with sudden intensity when the pressure on the artery was relaxed. Injections of adrenaline increased pain in febrile subjects but not in normal controls, and inhalation of amyl nitrite temporarily relieved it. Observations on the cerebro-spinal fluid showed that there was no relation between headache and a rise in the fluid pressure *per se*. In some cases jugular pressure relieved the pain, even though the pressure of the spinal fluid was increased; but after removal of some fluid jugular pressure might increase the headache though the fluid pressure did not rise. In other cases the effects were equivocal. The effects of compressing the extracranial arteries were also studied. Finally the authors concluded that in febrile headache pain was due to distension of the walls or perivascular tissues of dilated cranial arteries; in some cases the stimuli arose from intracranial arteries, in others from extracranial arteries, and in others again from both.

Pain in the eyes was found to be clearly related to vascular disturbances, but it appeared to be independent of cranial vascular changes. There was no definite relation between ocular tension and ocular pain; but the conclusion was reached that the basis of the pain was some undetermined vascular disturbance within the orbit. One would expect this to be the most likely explanation, in view of the congestion and tenderness of the eyeballs in dengue and sandfly fever. The pain on movement of the eye muscles is worth further investigation.

[1] *Clinical Science*, Volume VI, 1946, Numbers 1 and 2.

SYMBIOTIC CUTANEOUS GANGRENE.

DURING recent years an increasing number of articles have been written on a condition which has been chiefly noted by surgeons and dermatologists, namely, a spreading subacute or chronic gangrene of the skin and subcutaneous tissues. The bacterial flora has been varied, though the joint association of streptococci and staphylococci has been notable. Cases have occurred in the region of operation wounds where no previous infection has been known to exist; descriptions of such have been published from time to time in this journal. An article has been published on "Ulcus Phagedenicum Cutis" by A. Dostrovsky and F. Sagher from the Rothschild-Hadassah Hospital in Jerusalem.[1] These authors have collected data on fifty-eight cases, most of them observed during the last few years. It is evident from their description that only a limited proportion of the patients suffered from an acute and dangerous form of this condition since twenty-five of them were treated as outpatients only. The particular interest in the present article lies, not only in the careful observation of the cases, but also in the part of the world from which they

[1] *Archives of Dermatology and Syphilology*, October, 1946.

are reported. The problem of the so-called "desert sore", to mention one of its many names, was given much attention by Army medical officers during the Middle East campaigns and similar experiences were gained in the South-West Pacific Area. It is, of course, evident that by no means all of these infected states of the skin can be regarded as true symbiotic gangrene, but it certainly is interesting that these observers in Palestine have noted that the latter condition is common there.

They describe the ulcer as beginning in a pustule containing blood-stained fluid and with a blush red areola. Even at this early stage there is evidence of skin destruction and the edges are undermined. There is sometimes an offensive odour. In the acute cases rapid progression may occur by extension or by corridors undermining the intervening skin which only later becomes necrosed if at all. The location of the lesions is in the opinion of the authors dependent upon damage. Consequently, situations where injury and pressure are likely to occur are particularly vulnerable. This explains the great frequency of the ulcers in the lower part of the leg, especially over the tibia and the malleoli. The authors remark that the age distribution is in their case somewhat influenced by the relatively large number of young adults who have entered Palestine as immigrants. Most of their patients were under the age of forty and the majority were males. In many cases, trauma was reported by the patients, but in others no such cause was apparent. Bacteriological investigation established a joint infection by hæmolytic streptococci and *Staphylococcus aureus* in nearly half the cases; anaerobic streptococci were found in a few. These findings are in accordance with the other published work on this condition. Histological studies show evidence of an extensive inflammatory process, but do not shed light on the reason underlying the acute spread.

These writers point out that the so-called "desert sores" or "tropical ulcers" belong to several groups. They refer, for instance, to the type in which fusospirillary infection is present; they mention particularly the severe pain which is frequently associated with this type. The importance of this variety in some of the Pacific islands has been pointed out by contributors to this journal.

The use of sulphonamide drugs has proved very beneficial and perhaps we may hope that the severer forms of this depressing condition should now no longer occur. If, however, it is true that a spreading phagedenic infection is not uncommon in this part of the Middle East, it is perhaps a tribute to the early and thorough treatment of skin ulcers occurring in the armed forces in this part of the world. We understand that bacteriological investigation in Australian medical units in the Middle East showed that a number of streptococcal types were involved in the production and maintenance of these ulcers and numbers of cases were observed in which it was not easy to stimulate healing. Quite apart from lesions of this type are the rare cases of symbiotic gangrene which arise obscurely after simple and uncomplicated operations; still, these should be coped with successfully now, particularly if it is remembered how serious a condition may arise if prompt and effective measures are not taken.

SYMPTOMS IN CARDIO-VASCULAR DISEASE.

THE psychosomatic outlook on ill-health in some ways reminds us of combined operations in the military sense, though the ideal balance may be difficult of attainment. The medical practitioner is also faced with the difficulty that, whereas certain of his data may be correct to a high degree of accuracy, others are not easily susceptible to convincing measurement. George A. Wolf, junior, and Harold G. Wolff have published the results of a research into the nature of certain symptoms associated with cardio-vascular disorders, in which they have tried to pay due attention to both physical and mental factors.[1] They have succeeded in avoiding most of the inaccuracies of assessment of the mental factors by observing the physical changes demonstrable in persons living over again in their confidential interview with the doctor those elements of their daily life that were potent causes of psychic unrest. In order to do this a ballistocardiograph was used to estimate cardiac output, stress being laid upon the percentage changes observed over the average daily resting level. Spirometer readings were also taken, and oxygen consumption, minute ventilation, vital capacity and other determinations were made. Before any attempt was made to assess the symptoms of patients, two trained observers were used as test subjects for many months. Daily readings were made, standard exercise tests were applied, and then further readings were taken, after which details of the day's events were discussed and recorded. Psychological material was obtained by free association, by dream discussion and analysis, by description of behaviour, and by other appropriate methods. In this way a detailed account of the daily life of the subject was obtained, and experience was gained in the variations which might occur in the performance of the cardio-vascular system under differing conditions of life. Observations were then carried out on patients complaining of various symptoms referable to the circulatory system, and by a similar though of course less prolonged and laborious procedure, an evaluation was attempted of the various factors which might affect the performance and behaviour of the cardio-vascular power unit. The authors point out that they have placed emphasis on those low-grade stresses which are an integral part of everyday living. It is such strains, as they justly remark, which constitute the core of the bedside problem, rather than the well-known responses to the greater crises of life. The symptoms they investigated were dyspnœa, palpitation, pain believed to arise in the heart, fainting and giddiness, and fatigue. Complete clinical histories were taken from each patient, and the factors believed to be relevant, whether physical or psychological, were exposed by full investigation. Thus it was possible to arrive at a conclusion as to the possible importance of these factors, by studying the variations in the physical response to the different tests on a number of occasions. In discussing the general features of the results the authors begin by stating that the usual teaching in the literature is that anxiety has a more or less pronounced effect upon bodily functions. This approach tends to focus attention on the emotional aspects of the response to a situation, whereas it is the effect of the situation on the entire organism that is important to the physician trying to arrive at a just conclusion. The responses of the cardio-vascular system to excitement or stress are familiar, and the carefully made observations in this research have been employed to study to what degree such responses could be produced either by some contrived drama in the laboratory or by the feeling reactions to the internal drama of maladjusted persons. It was found that the latter reactions were much more intense in their physical effect. Such stresses as anger, guilt, rage, frustration and tension or anxiety were found to be potent in the production of palpitation with increased stroke-volume of the heart, of dyspnœa with inefficient pulmonary ventilation, of pain of the "cardiac" type, of giddiness and of fatigue. Where coronary arterial disease existed it was found that a feeling of defeat or desperation could produce pain. The authors found that there was a pronounced difference between persons with regard to the intensity and duration of such symptoms, as of course one would expect. It is known that certain persons do not appear to experience pain in cardio-vascular disease so readily as others, but whether the reasons for this are physical or psychological it is usually difficult to determine. In their summary the authors put the conclusions succinctly and well. Performance in terms of respiratory and cardiac function is costly, they point out, when the conditions of life and the reactions thereby excited are adverse. This high cost of performance may be manifest in the occurrence of cardio-vascular symptoms not solely due to structural disease, and impairment of the total capacity of the subject may result.

[1] *Psychosomatic Medicine*, September-October, 1946.

Abstracts from Medical Literature.

PATHOLOGY.

Relapsing Febrile Nodular Inflammation of Adipose Tissue (Weber-Christian Syndrome).

THIRTY-ONE cases have now been reported of the syndrome which was first recognized by Weber (1925) and described by him under the title "relapsing non-suppurative nodular panniculitis". To this designation Christian (1928) added "febrile". The literature contains reports of only four fatal cases, three of which were examined *post mortem*. A fourth fatal case of the Weber-Christian syndrome with post-mortem findings is reported by Henry Ungar (*The Journal of Pathology and Bacteriology*, April, 1946). Autopsy showed that the lesions were not limited to the *panniculus adiposus*, but extended also to adipose tissue in the thorax and abdomen. Histologically the initial lesion was distinguished by leucocytic stasis in the blood capillaries and focal suppurative inflammation in the adipose tissue. It was followed by the development of granuloma-like lesions which arose on the axis of the fibrous septa. The process terminated in scar-like formations or foci of foamy cell infiltration. The possible ætiology and the relationship between the different types of lesions are discussed.

Adrenal Hæmorrhages in Meningococcal Sepsis.

J. SCHWARTZ (*Archives of Pathology*, May, 1946) has observed adrenal hæmorrhages in five cases of fulminant meningococcal sepsis; the hæmorrhages began in the medulla and occurred by diapedesis. Generally medullary lesions were much more extensive than cortical lesions. In the cases observed inflammatory lesions were not found in the adrenal glands, nor was thrombosis of the adrenal veins found. Definite hyperplasia of the thymus and hypoplasia of the adrenal glands occurred, which is interpreted as explained by the seriousness of the syndrome. For historical reasons, the name "Waterhouse-Friderichsen syndrome" is, according to the author, not justified.

Mediastinal Chorionepithelioma in a Male.

A CASE of a twenty-six-year-old male with a large chorionepithelioma, primary in the mediastinum, presumably arising in a teratoma, is described by Oscar Hirsch, Stanley L. Robbins and John D. Houghton (*The American Journal of Pathology*, July, 1946). Serial sections of both testes, taken two millimetres apart, showed no gross or microscopic evidence of tumour. Gynæcomastia, a positive response to the Aschheim-Zondek test, and changes in the pituitary body resembling those seen in pregnancy were all present. Fourteen other cases of extragenital chorionepithelioma in males have been collected from the literature. The greatest incidence of these lesions is found, according to the authors, in the third and fourth decades of life. Gynæcomastia, or at least glandular hypertrophy, is frequently found in these cases in males, and in certain instances colostrum formation has been observed. Inasmuch as a primary tumour in the testes may be extremely small or may regress to microscopic size, metastasis from the testes may be considered and ruled out in all instances of supposed primary extragenital chorionepithelioma by a thorough examination of those organs, preferably by multiple sections examined microscopically.

Renal Lesions in Portal Cirrhosis.

J. H. BAXTER AND C. T. ASHWORTH (*Archives of Pathology*, May, 1946) have studied twenty-five cases of relatively uncomplicated portal cirrhosis to determine whether renal lesions were consistently present. Degenerative changes of the convoluted tubules and to a lesser extent of the loops of Henle, consisting of granular swelling with or without hydropic, vacuolar and fatty degeneration and hyaline droplet formation, were encountered to some degree in all cases. In some cases evidence of a more chronic form of renal injury was revealed, characterized by dilatation, atrophy and regeneration of the convoluted tubules. Glomerular changes consisting of periglomerular fibrosis and hyaline thickening of the basement membrane were observed by the authors, but these could not be definitely attributed to the cirrhosis. Albuminous precipitate was frequently observed in the glomerular spaces. Albuminuria was present in 60% of the cases, and this was attributed to renal parenchymal injury resulting in either an increase of glomerular permeability or a decrease of tubular function. A direct correlation was demonstrated between the activity of the cirrhotic process and the severity of the renal lesions. All degrees of tubular degeneration were observed in the patients with no jaundice as well as in those with jaundice, and the correlation between the activity of the cirrhosis and the degree of tubular injury was equally good in the two groups, although in general the severity of the tubular degeneration was somewhat greater in the group with jaundice. Although it cannot at present be determined with certainty, the authors believe that the renal changes described in these cases and in certain other types of cases in which hepatic and renal lesions occur simultaneously, may result not directly or indirectly from injury of the liver, but rather from the same factors which produce the injury of the liver.

Obliterative Cerebral Arteriosclerosis.

I. MARK SCHEINKER (*The American Journal of Pathology*, May, 1946) describes distinctive alterations of the smaller blood vessels as found in the brain of ten persons who had suffered from cerebral arteriosclerosis. This lesion is characterized by tremendous expansion of the intima, resulting in narrowing or complete obliteration of the vessel lumen. It is proposed that this process be designated as "obliterative arteriosclerosis", and considered as a special type of arteriosclerosis of small cerebral blood vessels. Emphasis is placed on differentiation from "hyperplastic sclerosis". Histological changes in the parenchyma of the brain, particularly the cortical grey matter, consisted of diffusely scattered, stripe-like, small, old and recent softenings secondary to the obliterative vascular lesions. A gross finding in the brain which was regarded as characteristic of "obliterative arteriosclerosis", was a granulated appearance of the cortical surface, due to numerous focal areas of glial scarring, often associated with stripe-like areas of softening involving the upper layers of the cortical ribbon. "Obliterative arteriosclerosis" may occur independently of arteriosclerotic changes of the major cerebral arteries.

Multiple Myeloma (Plasmocytoma) with Secondary Deposits in the Dura Mater.

ELIZABETH ARMSTRONG, J. STEVEN FAULDS AND M. J. STEWART (*The Journal of Pathology and Bacteriology*, April, 1946) describe two cases of multiple plasmocytoma, one in a woman, aged forty-seven years, in whom there were numerous medullary and lymph-glandular lesions and one large and one small deposit in the cerebral *dura mater*, the other in a male lunatic, aged seventy-seven years, in whom there were two massive and two smaller deposits in the cerebral dura and deposits in two ribs. In this case the dural tumours had penetrated completely through the whole thickness of the vault of the skull, while in the first case only the much thinned outer table remained over the larger growth. The authors concluded that the dural tumours were due to blood-borne metastasis and were not part of a multifocal primary systemic change. Only one similar case has been found in the literature.

The Leucopenic Factor of Exudates.

ACCORDING to Valy Menkin (*Archives of Pathology*, August, 1946), the leucopenia following the intravascular injection of an acid exudate, of pyrexin or of the leucopenic factor is accompanied by a trapping of apparently all types of leucocytes in the alveolar walls of the lungs, in the sinusoids of the liver, in the splenic pulp and perhaps in the marrow. This trapping of leucocytes offers a reasonable explanation for the mechanism of the leucopenia following the injection of the leucopenic factor. The trapping of leucocytes in the pulp of the spleen may serve to explain in a reasonable way the primary mechanism of the acute splenic tumour accompanying numerous inflammatory processes. Pyrexin, the leucopenic factor and necrosin seem to contain a common glycogen-inducing factor such that injection of any of these substances is followed by deposition of large quantities of glycogen in the hepatic cells and to some extent in the cardiac muscle fibres. The author is further investigating this factor.

Pathological Aspects of Atmospheric Blast Injuries in Man.

ACCORDING to Hilliard Cohne and Gerson R. Bisking (*Archives of Pathology*, July, 1946), the principal and characteristic lesion in eleven selected cases of atmospheric blast injuries was severe diffuse bilateral traumatic hæmorrhage of the lungs. All lobes of both lungs and all portions of a given lobe were involved in the hæmorrhagic process. Intense œdema and emphysema of the lungs occurred almost as frequently as did hæmorrhage. The emphysema was predominantly vesicular in type; the interstitial variety was noted only twice. The bronchioles, though perhaps not so vulnerable as the remainder of the pulmonary parenchyma, were also susceptible to the damaging effects of

the blast wave; the epithelium became loosened or detached from the *lamina propria*, erythrocytes, œdema fluid and leucocytes being extravasated into the injured bronchiolar wall. Pulmonary fat embolism was moderate in three and minimal in five cases. The authors regard this degree of fat embolism as neither of significance in the causation of the pulmonary lesions nor of influence in the clinical course of blast injury. Although the predominant damage of blast is intrapulmonary, none of the soft tissues or other viscera is absolutely immune to the destructive effects of the blast wave. In every case extrapulmonary lesions occurred in a variety of organs as small hæmorrhages, or very striking extrapulmonary lesions were found; in one case there was a "blow-out" perforation of the sigmoid colon; in a second, a massive hæmorrhage in the mesentery; in a third, a large horizontal tear and a massive hæmorrhage in the cerebellum; in a fourth, an extensive hæmorrhage in the wall of the stomach.

MORPHOLOGY.

Deep Lymphatics of the Spleen.

T. SNOOK (*The Anatomical Record*, January, 1946) has shown that, contrary to the most recent statements on the subject, deep lymphatic plexuses piercing the white pulp (lymphoid sheaths) are present in the spleens of the guinea-pig, mole, mouse, horse and monkey. In the guinea-pig, mole and mouse they follow the course of the white pulp arteries and open into lymphatic vessels at the hilus. In the horse they can be traced into trabecular and capsular lymphatic plexuses. These channels contain large numbers of lymphocytes, which should be considered in any analysis of the leucocyte-producing potencies of the spleen. Comparisons of arterial and venous blood alone would not give a true picture of splenic hæmatopoiesis in the animals listed.

Protrusion Mechanism of the Tongue.

G. A. BENNETT AND R. C. HUTCHINSON (*The Anatomical Record*, January, 1946) point out that according to most authors the genioglossus is specially concerned in protrusion of the tongue. Not until Bennett and Ramsay showed experimentally that part of the protrusion and some of the deviation of the tip of the tongue in certain mammals were functions of the intrinsic muscles did it become evident that more than one group of muscles was concerned in the complicated movement of protrusion. Furthermore, by means of these experiments it was possible to explain more satisfactorily than before why, in unilateral paralysis of the human tongue, a deviation of its tip to the paralysed side occurred on protrusion. However, these experiments did not determine the exact interrelationship of all of the functions of the extrinsic and intrinsic muscles of the tongue. The present investigation consists of a series of experiments performed on the whole and the split tongue of the dog, and attempts to explain the role of both the intrinsic and extrinsic muscles acting singly and together in protrusion of the tongue. It is shown that the protrusion of the tongue is due to the action of the genioglossi and the intrinsic muscles. The genioglossi pull the base of the tongue forward in the mid-line and fix its proximal two-thirds to the floor of the oral cavity, while the intrinsic muscles project it by means of an elongating process. The other extrinsic muscles are retractor muscles and can only alter the extent of the protrusion. In case the tongue is protruded by the action of the genioglossus and intrinsic muscles of one side, the weight or lag of the non-contracting half affects the direction of the protrusion in such a manner that the tip of the organ is contralaterally directed.

Quantitative Histology of Wallerian Degeneration.

M. ABERCROMBIE AND M. L. JOHNSON (*Journal of Anatomy*, January, 1946) survey the changes in the number of nuclei of nerves, excluding the perineurium and epineurium, during Wallerian degeneration, as seen in histological sections. The nuclei of different kinds of cells have, as far as practicable, been analysed separately. The authors have distinguished between nuclei within the Schwann tubes and nuclei in the endoneurium. The tubal nuclei have been separated into three groups according to size of the tube, and the endoneurial nuclei into connective tissue nuclei (fibrocytes and macrophages) and blood vessel nuclei (endothelial and smooth muscle nuclei). The authors state that the difficult and complicated history of the migratory macrophages cannot be adequately followed in their material. Evidence as to their multiplication and movement can, however, be got from vitally stained animals, and the authors will consider this in a subsequent paper. For the present, macrophage and fibrocyte nuclei are not distinguished in the endoneurium, nor macrophage and Schwann nuclei in the Schwann tubes. The changes in nuclear population during Wallerian degeneration are recorded up to 225 days of degeneration. The total population shows no change during the first three days, and then the growth curve shows a rise to peak population about eight times the initial population at about the twenty-sixth day, after which the curve falls, so that the population on the two hundred and twenty-fifth day is rather more than half that at the peak. The cells inside the Schwann tubes account for most of the population changes.

Human Pyramidal Tract.

A. M. LASSEK AND J. P. EVANS (*Journal of Comparative Neurology*, February, 1946) present a study of the pyramidal tract region in the spinal cord in an individual upon whom a unilateral hemispherectomy had been performed. They find that although there is practically complete degeneration of descending fibres in the pyramids of the medulla, the pyramidal tract area in the cord still contains many axones. Relatively more fibres are left intact in the cord area in the crossed or lateral than in the direct or ventral cortico-spinal component. Lesions of the pyramidal tract area of the spinal cord must therefore affect fibres other than those arising from the cerebral cortex of one side. Phylogenetically the pyramidal system progressively forms synapses with more and more segmental cells of the cord as the scale is ascended, and although it reaches its highest development in man, it appears that it does not occupy all the pyramidal tract area in the cord. It appears to be mixed with other fibres, and this is more pronounced in the lower regions of the cord. It is suggested that their destruction may account for some of the signs and symptoms of cord lesions in the lateral funiculus.

The Effect of Uterine Environment on the Skeleton.

E. L. GREEN AND M. C. GREEN (*Journal of Morphology*, January, 1946) state that when all or nearly all genetic variability has been eliminated, as by continued close inbreeding, there may still remain a large residual variability which must be associated with variable factors in the environment. The authors discuss the various factors of the embryonic environment that may cause significant variation in the developmental pattern, and in an investigation found no evidence for the effect of any of them on skeletal variability. The sources of non-genetic variation must be much more intangible than those yet tested. The investigation was carried out on a special strain of mice in which there exists a high amount of non-genetic variability in the axial skeleton, making the strain and the characteristics suitable for an attempt to estimate the effects of prenatal environmental factors on skeletal variability.

Fusion of Notochord to Neural Tube.

B. M. PATTEN (*The Anatomical Record*, July, 1946) records a rare embryological malformation in which there is local fusion of the notochord with the developing spinal cord. The author offers suggestions as to the possible resultant abnormalities at later stages which might have developed had the embryo been permitted to continue its development.

Regeneration of Nerve.

D. DENNY-BROWN (*Archives of Neurology and Psychiatry*, March, 1946) has investigated the factors which aid or hinder the growth of axones across the gap when there has been a break in continuity in a nerve trunk. So far this process has been imperfectly understood. The author finds that the perineurium and endoneurium of peripheral nerve are formed of specialized connective tissue cells of two types. One is a large flat cell of mesothelial type, seen typically in the perineurial lamellæ. The other is a specialized fibroblast. The Schwann cells play only a subsidiary part in the regeneration of nerve. The neural fibroblast is immediately activated by injury and then proliferates. It invariably accompanies regenerating nerve fibres, often preceding them, and ensheaths newly formed nerve fibres and bundles. The large flat mesothelial cells provide an outer perineurium. The uncontrolled migration of these mesoblastic cells is responsible for the traumatic neuroma. Dispersal can be prevented by the provision of an intact perineurial sheath. The fibrosis of suture lines and grafts is associated with previous activity of the neural fibroblast. Factors of importance in the production of collagen by these cells when once activated are ischæmia, tension and their aging without provision of nerve fibres. Finally the author finds that perineurium can be utilized for efficient repair of defects in nerve.

Bibliography of Scientific and Industrial Reports.[1]

THE RESULTS OF WAR-TIME RESEARCH.

During the war a great deal of research was carried out under the auspices of the Allied Governments. It has been decided to release for general use a large proportion of the results of this research, together with information taken from former enemy countries as a form of reparations. With this end in view, the United States Department of Commerce, through its Publication Board, is making a weekly issue of abstracts of reports in the form of a "Bibliography of Scientific and Industrial Reports". This bibliography is now being received in Australia, and relevant extracts are reproduced hereunder.

Copies of the original reports may be obtained in two ways: (a) Microfilm or photostat copies may be purchased from the United States through the Council for Scientific and Industrial Research Information Service. Those desiring to avail themselves of this service should send the Australian equivalent of the net quoted United States price to the Council for Scientific and Industrial Research Information Service, 425, St. Kilda Road, Melbourne, S.C.2, and quote the PB number, author's name, and the subject of the abstract. All other charges will be borne by the Council for Scientific and Industrial Research. (b) The following reports may be obtained in approved cases without cost on making application to the Secondary Industries Division of the Ministry of Post-War Reconstruction, Wentworth House, 203, Collins Street, Melbourne, C.1. Copies of these are available for reference in public libraries.

Further information on subjects covered in the reports and kindred subjects may be obtained by approaching the Council for Scientific and Industrial Research Information Service, the Secondary Industries Division of the Ministry of Post-War Reconstruction, or the Munitions Supply Laboratories (Technical Information Section), Maribyrnong, Victoria.

LAUGHLIN, ROBERT C. The therapeutic effectiveness of BAL solution against liquid PD, ED and $AsCl_3$ in eyes of rabbits. (Chemical Warfare Service. Medical Research Laboratory. MRL(EA) Report 28.) Off. Pub. Bd., Report, PB 9557. 1944. 14 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The eyes of unanæsthetized rabbits were contaminated with liquid PD, ED and $AsCl_3$ by the splash and blink method. The amount of PD used was 0·64 milligramme and the eyes were treated with several drops of 5% BAL solution after thirty seconds, three minutes and ten minutes. The amount of ED was 0·6 milligramme and 0·3 milligramme and the animals were treated after thirty seconds and two minutes with varying amounts of BAL solution. Controls were run on PD and ED with no treatment and with water irrigation. An application of 0·77 milligramme $AsCl_3$ was used and the eyes treated after thirty seconds with BAL solution. All of the eyes contaminated with PD and treated after thirty seconds or three minutes recovered completely. Only one out of ten treated at ten minutes recovered; the others had either perforated by the fourteenth day or were about to perforate. All eyes contaminated with 0·3 milligramme ED and treated at two minutes or less recovered. With a dose of 0·6 milligramme most of the eyes recovered completely, but one treated at two minutes and two treated at thirty seconds perforated. BAL had no therapeutic effect on eyes contaminated with $AsCl_3$. Tables are appended.

LEITCH, JAMES L., AND BAUER, VIRGINIA E. Oral toxicity of cyanogen chloride in water to rats. (Chemical Warfare Service. Medical Division Report 19.) Off. Pub. Bd., Report, PB 9423. 1945. 11 pp. Price: Microfilm, 50c. Photostat, $1.00.

Tests were made to determine the toxicity of cyanogen chloride in water to rats when administered by stomach tube. The results, presented in tabular form, indicate that the oral LD 50 of cyanogen chloride for rats is approximately 6·0 milligrammes per kilogram.

LEITCH, J. L., AND GINSBURG, T. H. Purification of water contaminated with chemical agents. The toxicity to rats of certain chemical agents in water. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 30.) Off. Pub. Bd., Report, PB 13675. 1941. 34 pp. Price: Microfilm, 50c.; Photostat, $3.00.

Study was made of the toxicity to rats of water contaminated with phosgene (CG), chloropicrin (PS), methyldichlorarsine (MD), diphenylchlorarsine (DA), diphenylaminechlorarsine (DM) and diphenylcyanarsine (CDA). There was no evidence that any of the agents produced any pathological changes under the conditions of these experiments. Tables are included.

LONGCOPE, W. T., *et alii*. A test programme on the physiological actions of PF_3 in normal individuals and in patients with *myasthenia gravis*; bimonthly progress report 3, January 15, 1946. Off. Pub. Bd., Report, PB 13741. 1946. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Contents: 1. Physiological effects of PF_3 on human subjects. 2. The effects of intraarterial injections of choline chloride and reducing agents. 3. Clinical observations on patients with *myasthenia gravis*. Includes tables.

LYON, GEORGE M., *et alii*. An evaluation of cobalt nitrate as an antidote for cyanide poisoning. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 84.) Off. Pub. Bd., Report, PB 11382. 1943. 25 pp. Price: Microfilm, 50c.; Photostat, $2.00.

This report describes the procedure and results of tests to determine the value of cobalt nitrate as an antidote for cyanide poisoning. Tables present test data which show that cobalt nitrate decreases the mortality due to KCN poisoning in rats, rabbits and goats. The toxicity produced by HCN is reduced following the prophylactic administration of cobalt nitrate orally or subcutaneously in the animals studied.

MACE, DON L. Experimental treatment of phosgene poisoning. Expiration against positive pressure. The sedative effects of sodium barbital, sodium bromide and potassium bromide. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 32.) Off. Pub. Bd., Report, PB 13674. 1942. 17 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The treatments cited above were applied to goats exposed to phosgene with the intention of curtailing the formation of pulmonary œdema. Neither of these measures decreased the death rate. Bibliography, tables and a diagram are included.

MCNAMARA, B. P., HARRISON, A. E., AND NEVILLE, G. A. Cobalt nitrate-ascorbic acid combination as an antidote in HCN poisoning. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 103.) Off. Pub. Bd., Report, PB 11442. 1943. 18 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The toxicities of a 10% solution of cobalt nitrate and of a solution containing 10% cobalt nitrate *plus* 2·7% ascorbic acid were determined by intravenous administration to rabbits. The LD 50 of cobalt nitrate was about 32 milligrammes per kilogram for the former solution, while the LD 50 for cobalt nitrate in the latter solution was about 68 milligrammes per kilogram. When these solutions were administered to rabbits, immediately before a five-minute exposure to HCN, they were of similar value in counteracting the cyanide toxicity. The LD 50 for such an exposure in untreated animals was about 0·5 milligramme per litre, whereas the LD 50 of HCN in the treated animals was about 1·0 milligramme per kilogram. However, the concentration-mortality graphs developed from these experiments indicate a 95% mortality could be expected from 2·0 milligrammes per litre of HCN after a five-minute exposure regardless of prophylactic treatment. Tables and charts are included.

MCNAMARA, B. P., HARRISON, A. E., AND GUBERMAN, E. A. The comparative effects of greasy and non-greasy BAL ointments, with and without lavage, on rabbit eyes contaminated with liquid mixture of Lewisite and mustard. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 98.) Off. Pub. Bd., Report, PB 11436. 1943. 16 pp. Price: Microfilm, 50c.; Photostat, $2.00.

Several ointments containing BAL were used as treatment agents in rabbit eyes contaminated by a 1:1 (volume to volume) liquid mixture of Lewisite and mustard. The effect of lavage following such treatment was also studied. It was found that when 0·9 milligramme or more of the mixed vesicants was applied to the cornea, none of the treatment agents were successful. Irrigation did not improve the value of these treatments. Most eyes receiving such contamination became non-functional. When 0·29 milligramme of the mixed vesicants was applied to the eyes, the resulting damage was much less severe. On the six days during which this group of animals was observed there was no significant difference between the treatment techniques and lavage did not seem to alter their value.

MCNAMARA, B. P., *et alii*. The value of ferrous sulphate and cobalt-cysteine combinations in HCN poisoning. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 86.) Off. Pub. Bd., Report, PB 11384. 1943. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Prophylactic doses of thirty milligrammes per kilogram of ferrous sulphate failed to lower the mortality produced by HCN. The administration of this iron salt appeared to lower the resistance of rabbits to poisoning by HCN. Various cobalt-cysteine combinations, containing the amount of cobalt nitrate which alone was previously found to be

[1] Supplied by the Information Service of the Council for Scientific and Industrial Research.

beneficial in cyanide poisoning, were valueless as prophylactics in HCN poisoning, when given orally or intravenously.

MARQUAND, C. B., *et alii.* Evaluation of solvents for removal of liquid mustard from skin. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 83.) Off. Pub. Bd., Report, PB 11381. 1943. 16 pp. Price: Microfilm, 50c.; Photostat, $2.00.

Tests were made to evaluate the usefulness of solvents to arrest mustard burns on skin. The best solvents, such as hydrogenated vegetable oil, linseed oil and ether, effected a reduction of 70% in the injured areas due to mustard. Absorbents were previously shown to be capable of reducing the injured area 80%. All of the solvents investigated except two, chlorcosan and carbon disulphide, were instrumental in effecting reductions in the mustard-burned areas of rabbits. Dry swabbing was more effective in reducing injuries than the usually recommended decontaminants such as kerosene, gasoline, or soap and water. Despite the fact that the technique employed showed the maximum efficiency for the solvents, the personnel treating the animals were injured due to spreading of mustard by the solvents. Tables present test data.

MARQUAND, C. B., *et alii.* The use of absorbents in the prophylactic treatment of liquid mustard burns on rabbits. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 80.) Off. Pub. Bd., Report, PB 11377. 1943. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

A series of 27 dry absorbent materials were used on rabbits 15 minutes following application of 13 milligrammes of mustard. These gave a variety of results, some preventing as much as 80% of the damage done by mustard, others showing little value. Solvents have shown less effectiveness than absorbents. Of all materials studied, magnesium oxide *plus* HTH, calcium carbonate, filter paper pulp with charcoal and plaster of Paris powder showed the most effective removal of mustard.

MARQUAND, C. B., *et alii.* The use of hyperol (urea-hydrogen peroxide $CO(NH_2)_2.H_2O_2$) followed by wet dressings in the first-aid treatment of liquid Lewisite burns of rabbits. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 58.) Off. Pub. Bd., Report, PB 11189. 1942. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Fifty-four animals were burned with liquid Lewisite and treated with 20% or 30% hyperol and wet dressings of 3% H_2O_2 or 10% hyperol. The combined treatment showed certain slight advantages over the use of hyperol alone, and equally as many disadvantages. In all cases, the treatment was not as effective as 8% H_2O_2. On the whole, wet dressings of 3% H_2O_2 or 10% hyperol did not significantly improve the therapeutic value of either 20% or 30% hyperol as a treatment for liquid Lewisite burns. A bibliography is included and tables are appended.

MAZUR, ABRAHAM, AND BODANSKY, OSCAR. The mechanism of in-vitro and in-vivo inhibition of cholinesterase activity by PF_3 in the rabbit, monkey and man. (Chemical Warfare Service. Medical Research Laboratory. MRL(EA) Report 25.) Off. Pub. Bd., Report, PB 9554. 1944. 69 pp. Price: Microfilm, $1.00; Photostat, $5.00.

Experiments were made to investigate the mechanisms involved in the decrease of cholinesterase activity by PF_3 and to determine whether this decrease was reversible. Results obtained indicate that PF_3 acts on cholinesterase so as to form a difficult reversible or perhaps even irreversible substance. The rate of restoration of serum cholinesterase to normal activity after exposure is very slow and is similar to the rate of synthesis of serum protein. Human serum cholinesterase is very sensitive both *in vivo* and *in vitro* to inhibition by PF_3. Exposures of man to CT values ranging from 165 to 290 result in marked decreases of the serum cholinesterase activity to 1% to 5% of pre-exposure values. Systemic symptoms at this range of exposures are slight, but analogy to experiments with the Rhesus monkey indicates that somewhat higher exposures may inactivate the brain and other tissue cholinesterases and result in marked systemic symptoms. Tables and a bibliography are included.

MAZUR, ABRAHAM, AND BODANSKY, OSCAR. The regeneration of cholinesterase activity in PF_3 poisoning and the effect of injected cholinesterase preparations in normal and PF_3 poisoned animals. (Chemical Warfare Service. Medical Division Report 35.) Off. Pub. Bd., Report, PB 9524. 1945. 34 pp. Price: Microfilm, 50c.; Photostat, $3.00.

Rabbits injected intravenously with a single dose of 0·3 milligramme per kilogram PF_3 showed subsequent depression of the cholinesterase activity of serum, red cells and brain. The serum cholinesterase activity returned to normal in five days, the red cells cholinesterase activity was restored to normal in ten days, and the brain cholinesterase activities attained normal values one to two months after the injection. The intravenous injection of large amounts of potent horse serum or electric eel cholinesterase preparations into rabbits and monkeys before or after the injection of PF_3 raised the serum cholinesterase activity, but did not affect the red cells and brain cholinesterase activity. No amelioration of symptoms or protection from lethal doses was obtained.

NAUDAIN, GLENN G., *et alii.* Use of carcasses of goats, contaminated with L, for food. (Chemical Warfare Service. Medical Research Laboratory. MRL(EA) Report 24.) Off. Pub. Bd., Report, PB 9553. 1944. 11 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Ten milligrammes per kilogram of L were applied to the shaved skin area on the gluteal region of the right hind leg of a goat. The goat died within 24 hours. Post-mortem examination was made and tissues collected for arsenic analyses and feeding tests. A second goat, exposed to L vapour for eight minutes by means of a beaker-cup arrangement held over the animal's nostrils, died within eighteen hours. Post-mortem examination was made and tissue collected for arsenic analyses. Results indicate that the carcasses of goats burned severely with liquid L may be used for emergency food purposes if the lesion and surrounding area of muscle tissue are trimmed to a depth of three to five inches. The flesh of a goat killed by inhalation of L vapour contains only non-toxic traces of arsenic and may be freely used for food. Tables and a bibliography are included.

ROWELL, WILFRED J., *et alii.* Tolerance of the mustard injured human eye for 5% sulphathiazole ophthalmic cream. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 100.) Off. Pub. Bd., Report, PB 11438. 1943. 5 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Results of tests presented in tabular form show that the right eyes of all ten patients were so severely irritated from 5% sulphathiazole cream that in fifteen to thirty minutes afterwards they require 0·5% "Pantocain" solution for the relief of pain. No permanent damage resulted, but it is concluded that the sulphathiazole cream is unsatisfactory for treatment of conjunctivitis caused by exposure to mustard gas vapours.

SILVER, S. D., MCGRATH, F. P., AND KRACKOW, E. H. Hydrocyanic acid, LC 50, for rats exposed two minutes. (Chemical Warfare Service. T.R.L.R. 22.) Off. Pub. Bd., Report, PB 11550. 1944. 8 pp. Price: Microfilm, 50c.; Photostat, $1.00.

Ten groups of ten rats each were exposed to varying concentrations of HCN for two minutes. Results presented in tabular form indicate that the LC 50 of hydrocyanic acid for rats exposed two minutes is 1·1 milligrammes per litre.

SILVERBLATT, ETHEL, *et alii.* Preliminary experiments on the toxicity of arsine (AsH_3) to rabbits by inhalation. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 29.) Off. Pub. Bd., Report, PB 13676. 1942. 60 pp. Price: Microfilm, $1.00; Photostat, $4.00.

A method was set up for gassing rabbits with arsine, and the pathology of the animals was studied. The agent caused a gross amount of erythrocyte destruction and pathological conditions of the liver, kidney and spleen. Other organs appeared normal. The absolute drop in blood count was approximately directly related to the concentration of arsine to which exposed. The time of appearance of hæmolysis and hæmaturia as a function of the arsine concentration was studied. Tables, diagrams, graphs and photographs are included.

WEXLER, JACK, *et alii.* Evaluation of alkaline therapy in WP burns. (Chemical Warfare Service, Edgewood Arsenal. Medical Division Report 25.) Off. Pub. Bd., Report, PB 9517. 1945. 12 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The assumption that alkali is beneficial in treatment of WP burns because it might neutralize the acids formed in the combustion of WP on skin is not borne out by the results of the experiment reported herein. On the contrary more favourable results were obtained with acid treatment in the form of pyruvic acid gel.

WILSON, CATHERINE E., *et alii.* The control of experimental pneumonia with penicillin. (Chemical Warfare Service. Medical Division Report 42.) Off. Pub. Bd., Report, PB 9527. 1945. 18 pp. Price: Microfilm, 50c.; Photostat, $2.00.

The object of this investigation was to test the effect of penicillin administered as an inhalant and by subcutaneous and intramuscular injection upon pneumococcal pneumonia produced experimentally in small animals. Results proved that the inhalation of penicillin aerosols by rats is as effective in reducing their mortality from experimental lobar pneumonia as is the administration of the agent by injection. Clinical trials are recommended by this report. Tables and a bibliography are included.

GILPIN, BURHL B., JUNIOR. Treatment of conjunctivitis and laryngitis due to exposure to mustard vapour. (Chemical Warfare Service. Medical Division. MD(EA) Memorandum Report 38.) Off. Pub. Bd., Report, PB 13668. 1941. 6 pp. Price: Microfilm, 50c.; Photostat, $1.00.

The object of this paper is to describe a small series of cases of mustard conjunctivitis which occurred at Edgewood Arsenal, together with their treatment and prognosis. The use of sodium sulamyd is discussed.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association and of the New South Wales Branch of the Australian Dental Association was held on September 26, 1946, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. A. C. THOMAS, the President, in the chair.

Dento-Facial Deformity and Nasal Obstruction.

DR. R. H. BETTINGTON read a paper entitled "Nasal Obstruction in Children and its Relation to Dental Development and General Health". This paper was published in the issue of February 1, 1947, at page 134.

DR. A. THORNTON TAYLOR read a paper entitled "Dento-Facial Deformities Associated with Nasal Obstruction". This paper was published in the issue of February 1, 1947, at page 135.

DR. R. A. R. GREEN, in opening the discussion, congratulated those who had arranged the meeting on their choice of a subject of such interest to both professions. Dr. Green said that the point he had found outstanding in Dr. Bettington's paper was the remark of Hastings and James, which had been quoted, to the effect that adenoids did not cause nasal obstruction. Dr. Green agreed with them. He said that the enlargement of the adenoid tissue was usually secondary to some other condition causing nasal obstruction, such as infection. With another remark, to the effect that mouth breathing did not cause dental malocclusion, Dr. Green agreed. It was not the mouth breathing that caused deformity of the teeth or jaw structure, but the deformity of the jaw itself which led to mouth breathing, the deformity arising first from some chronic nasal obstruction. Dr. Thornton Taylor had given those present food for thought. Dr. Green himself thought that pædiatricians must take some of the blame for the fact that patients were not instructed properly after rhinological treatment, because often the rhinologist had been acting only as a helper of the pædiatrician, who had not always seen that the patient followed up the rhinological treatment by adequate after-treatment. An extenuating circumstance was that commonly these children were under care at a fairly early age—earlier than the age at which the mechanical treatment mentioned by Dr. Thornton Taylor was begun (about eight years). Some pædiatricians felt that the "springing" of arches could not be carried out on the bone and tooth structure of the first dentition. Again, it was not easy to teach a young child exercises or to have him persist in forced breathing. That was Dr. Green's apology. On the question of heredity or hereditary deformities, Dr. Green said that he felt such a concept to be unlikely. Nature generally tended to produce the best form for function. Dr. Green thought that sometimes sight was lost of the fact that children were quite early infected in their own homes. The mouth breather with chronic nasal infection was common. One looked round the child's home, and found that the mother or father had chronic nasal infection. The children were early infected and reinfected, the nasal mucous membrane became thickened and the children were forced to breathe through the mouth; the dental arches became narrowed, and the clinical picture resembled that of the parent. The condition was probably acquired and not hereditary. Referring to treatment, Dr. Green said that the question had been approached from different angles. The cases illustrated by Dr. Thornton Taylor were in fact the end results of various wrong doings. The thumb-sucking child did more than force back his lower jaw; the thumb was inserted into the mouth, the terminal joint pressed against the palate was pushed up by the tongue, and in certain cases the result was the forcing up of the palate, the pulling in of the sides of the dental arches, narrowing of the nostrils and so on. Finger sucking was not nearly so dreadful; the thumb went in and cocked up against the palate, whilst the finger went in and straight back. Dr. Green said that the results of all these habits depended on the state of the bone. He did not believe that in the case of a normal, healthy child thumb or dummy sucking would have any effect on the form of the jaw. When an abnormality like rickets was present, then the condition was one in which the bone was easily pushed into various wrong positions. Van Rees, writing on orthodontia, had pointed out that the rhachitic condition which was obvious in the long bones of children was also found in the mandible and maxilla. These rhachitic conditions could be such that the bone was easily deformed. Not only were the maxilla and the nasal arch easily deformed, but so also was the mandible, and the form of the mandible could be altered in rickets by muscular action. From this point of view, the important matter was prevention. In that regard, the feeding habits of the child were of the greatest importance. First of all, the artificially fed child was the one who was likely to suffer from rickets. If one watched a normal child feeding at the breast, one found that the child took an appreciable amount of breast tissue into the mouth. The lips had a definite function, the lower jaw entered the picture with a munching sort of action, and the whole mouth was stretched. In the early days of life, the function of the mouth was purely that of a sucking organ. When these children fed, the filling up of the mouth pushed the cheeks out and the pull of the muscles in sucking was outwards; the result was a pulled out dental arch. The artificially fed child usually had a bottle with a narrow teat, and the effect was the same as that of thumb sucking. Muscular action in suction was then downward and somewhat inward. This tended to narrow the dental arch and forced the palate up. That effect, again, occurred much more easily in rhachitic children. With the forcing up of the palate, various changes occurred in the nasal floor. The palate of a young baby was a low, broad structure; that of a child badly fed and badly handled was narrow and highly arched. With the thrusting up of the palate one found that the nasal septum was deviated; this was inevitable, because bones had to grow. In the young baby the inferior turbinates were close to the floor of the nostril. If the palate was thrust up, then occlusion of the nose would follow. If infection was superadded, then the child must become a mouth breather. Dr. Green said that from the discussion some useful lessons could be drawn. It seemed to him that the time was close when each of the hospitals dealing with children should have attached to the staff an orthodontist, who could advise and help in the prevention of the more serious deformities. If those present took away with them that idea, the meeting would have done some good.

DR. G. C. HALLIDAY spoke defending the attitude of the rhinologists in these cases, particularly as he had been one of those responsible for the choice of this subject. He said that the rhinologist was consulted by parents who brought him their child suffering from nasal obstruction, and on examination it was frequently found that there was no post-nasal adenoid tissue, as generally both the tonsils and adenoids had been adequately dealt with, and the obstruction was due to the maldevelopment of the upper and lower jaws causing a deflection of the septum and a disuse of the dilator muscles of the nose. The rhinologist would then ask whether the child's dentist had suggested a consultation with an orthodontist, and frequently the answer was "no". All rhinologists encountered many such cases. Dr. Halliday joined issue with Dr. Thornton Taylor on the question of blame attached to the rhinologists; 95% of such patients as mentioned above had had their tonsils removed by surgeons other than rhinologists, and by the time the rhinologists saw them, the deformity of their mouth and nasal structures was well established. Unless the dentist, pædiatrician and rhinologist worked in close cooperation, the problem of these children could not be overcome. It was the duty of the dentist to note if the child was a mouth breather, and then, if the teeth and jaws were in good order, he should send the patient to a rhinologist. The rhinologist on his part, seeing such patients, should make sure there was no abnormality in his department and send the child for orthodontal treatment if required; this should be done before the age of twelve years, as the palate and alveoli were very malleable bones at this stage and great help could be afforded in these cases. Dr. Halliday had patients suffering from allergic rhinitis unable to breathe through their noses, and once they commenced mouth breathing the upper lip became patulous and gave no support to the teeth. Malocclusion of the teeth could best be prevented by doctors and dentists working in complete cooperation and trying to prevent the establishment of early faults.

MR. E. B. GREEN referred to screens, examples of which had been passed round for inspection by Dr. Thornton Taylor. Mr. Green said that he had had many discussions with Dr. Taylor on the subject, and was glad that the screens had been exhibited. The first man to bring these oral screens before the dental profession was H. O. Dickin, who had written a thesis called "Oral Screens in the Treatment of Certain Dental Irregularities", and this was read before the Royal Society of Medicine in 1934. Dickin advocated the construction of these oral screens, and made a good many wider claims in regard to their value than the mere correction of dental irregularities. He stated that it would be found that the use of the oral screen would be followed by an increase in the strength of the lips, an

expansion of the dental arches and also of the true bone of the face, an increase in the height of the bite, a reduction in the incidence of dental caries, a greater resistance to infections of the air spaces of the head, an improved peripheral circulation of the blood throughout the whole body, a brightening of the mentality due to the better aeration of the air sinuses, more tranquil sleep, an increasing harmony of the features not confined to the dental arches, and a surprising improvement in the whole physique. Mr. Green said that there was something in the claims made for oral screens; he had had experience with them. He agreed with Dr. Halliday that a good deal of cooperation was necessary between the rhinologist and the dentist. He thought that the average member of the dental profession had not paid much attention to the mouth breathing; it was one of those spheres of activity in which they might take more interest. If some of their child patients suffered from these obstructions, the parents should be informed. After the child had been taken to the rhinologist he should be referred back to the dentist for the institution of good breathing habits by the use of screens or plasters. The screens were easy to construct; any dentist could make them. Children wore them easily and rapidly, and once they had formed the habit of wearing them at night, they were loath to do without them.

MR. OWEN S. B. HOGG said that no reference had been made to the function of the nostril. In many cases of obstruction, if the nostril was held out, the intake of air was sufficient. He used to make small vulcanite caps to hold the nostril out which proved very helpful in establishing nasal breathing. Mr. Hogg said that he did not altogether agree with what Dr. Thornton Taylor had said about the upper lip; he thought that the cause of mouth opening was that the lower jaw, at rest, instead of just touching the upper teeth, dropped down and back. Mr. Green had referred to the introduction of oral screens in 1934. Sir Frank Colyer had invented a screen in about 1912. Mr. Hogg had made such screens, but had not found them very satisfactory in practice. They were made of wire. Referring to Dr. Thornton Taylor's use of forced breathing, Mr. Hogg said that he was a little late; the man who originally urged that was not a dentist or doctor, but Sandow. Sandow advocated that the child should ride a bicycle or skip as much as he could with the mouth closed. Mr. Hogg had used the method in the treatment of children, with good results. The point was to establish normal function. If normal function (nasal breathing) could be brought about, some abnormal conditions and nasal obstructions would disappear. In conclusion, Mr. Hogg thanked the speakers for their interesting papers.

MR. C. J. GRIFFIN said that he had been interested to hear the views of the speakers on the subject of heredity, and particularly on how much heredity might contribute to the ætiology of the conditions that had been discussed. It seemed that throughout the primates there was a trend towards an increase in the cerebral part of the skull and a decrease in the facial part. This tendency was emphasized in *Homo*, in whom it was found reflected, as far as the facial part was concerned, in the width between the maxillary cuspids—in other words, from the palatal side of the socket of the cuspid to its fellow of the opposite side. In Piltdown man this measurement was in the neighbourhood of 40 millimetres, in Neanderthal man about 27 millimetres and in Bronze Age man about 22 millimetres; it was probably much less in the average modern European. In addition it was also found that deflection of the nasal septum was rare in the primitive type; Mr. Griffin believed that Larneck in association with Burkitt had found it once in fifty Australian aboriginal skulls, whilst in the European it was very common—so common as to be almost considered normal.

Mr. Griffin said that it had been his experience, in the examination of numerous skulls, to find that the most severe deflection of the septum was concentrated where the vomer articulated with the anterior part of the maxilla. The main consequence of this deflection was that the *septum nasi* on the side of the deflection pressed against the middle concha, which in turn hypertrophied and helped to occlude the ostium of the maxillary sinus on that side. Mr. Griffin believed that this set of conditions was the predisposing cause of many infections of the nasal cavities and accessory air sinuses. Mr. Griffin went on to say that the articulation to which he had referred was formed by processes of the premaxillæ, which were first observed by Rambaud and Renault, and which they called the subvomerine processes of the premaxillæ. Fawcett later identified them and called them the infra-vomerine processes of the premaxillæ. Jarmer also identified them as separate centres of ossification of the premaxillæ, and termed them individually the *"processus Stenionani"*. Valvis and Cadenat did not agree that they were separate centres of ossification, and termed them the medial palatal processes of the premaxilla; on this contention they were supported by Augier. Adolf Schultz, the Carnegie embryologist, had also observed asymmetry of the *alæ nasi* in white fœtuses. Mr. Griffin said that it had been his good fortune to observe the asymmetry of these processes, which in early ontogeny (40 millimetre stage) were located in the centre of the palate, but in later ontogeny (150 millimetre stage) were seen to project in the nasal fossæ and articulate with the vomer. He believed that deflection of the nasal septum was due to the asymmetry of these processes, which were placed in such a position as to suffer from any asymmetry of the facial bones. The homologizing of these processes would involve a description of the relationship of certain bones through mammalia, reptilia and pisces, and Mr. Griffin did not propose to discuss that question. The observations that he had made represented the result of three or four years of study of the problem.

DR. D. G. CARRUTHERS said that he thought it would be wrong not to accept the challenge of Dr. Thornton Taylor. He strongly refuted the statement that the parents of affected children were not carefully instructed about breathing exercises after operations for the removal of tonsils and adenoids. Dr. Carruthers could speak for himself, and he thought that his colleagues did the same as he did. The parents of the children were given full instructions. Dr. Carruthers usually had the child stand in front of him in the surgery and do some simple exercises; he showed the child what to do, and made notes at the time, and at the child's next visit asked questions on what had been done. Dr. Thornton Taylor might have been misled, because the cases coming within his practice were probably failures after rhinological treatment in which adequate development was absent and nose breathing was impossible. Other rhinologists and most medical practitioners would agree with him (Dr. Carruthers) that it was one of the greatest sources of satisfaction to the surgeon who removed a child's tonsils and adenoids to have the parent return two or three weeks later and say what a change had been noticed in the child, who was breathing well, doing well and sleeping naturally, and had adopted the habit of nose breathing in his sleep without obturators. Another point to which Dr. Carruthers wished to refer was the statement that adenoids did not cause nasal obstruction. He was certain that they did. Perhaps they did not always do so mechanically, but they could do it that way. In other cases they predisposed to and kept up nasal infection. The good results after adenoids had been removed were so speedy that it was obvious that the adenoid tissue was causing obstruction and having some effect on the palatopharyngeal musculature. Dr. Carruthers thought that Dr. Bettington had had a difficult task in his paper, because he had to mention, as well as the various aberrations that could be corrected relatively simply, those irregularities that were difficult to correct. There were, for example, children with allergic trouble which came not only in sensitized mucous membrane; but there were also amongst those children many who required some form of psychological treatment. The psychological factor was very deep; it was present in the parents as well as in the child, and in the environment. One did what was necessary and the parent was pleased with the result; then later the child returned, as badly affected as ever. It was obvious that the child's condition was allergic in origin, due to some disturbance at home or to some health upset or change in the weather. The mode of correction in such cases was very difficult, and as yet no obturator to fit these circumstances had been found. In conclusion, Dr. Carruthers thanked the speakers for their papers. He said that though he had risen in defence of the rhinologists, he was still willing to learn, and he would remember more of the orthodontist's aspect from having attended the meeting.

Dr. Bettington, in reply, said that he had been most appreciative of Dr. Thornton Taylor's paper, which gave evidence of a great deal of trouble, thought and work.

Dr. Thornton Taylor, in reply to Dr. Carruthers's remark that the orthodontist saw the rhinologist's failures, said that that might be so; it was logical. The orthodontist's two main worries concerned breathing and oral hygiene, and in efforts to educate patients he spoke seemingly millions of words yearly on these subjects. Most patients were good, intelligent children, who did not need continual prompting; some children, however, while good, were unable to cooperate. Sometimes the parents were intelligent and keen, but the children would not cooperate. Dr. Thornton Taylor appreciated the idea of cooperation between the three specialties. He had for a long time felt helpless over some of these rhinological problems. He had referred many children back for an opinion on the

tonsils and adenoids, and had on occasion been told that their removal was not necessary, so that he was thrown back on his own resources. Dr. Thornton Taylor thanked members for their discussion and was particularly appreciative of Dr. Green's well-balanced and broad comments on this subject.

DR. J. V. HALL BEST proposed a vote of thanks to the speakers, which was carried by acclamation.

Dr. Thomas, from the chair, thanked those present for their attendance. He said that the meeting had been successful from the point of view of both the dentists and the medical men present.

THE AUSTRALIAN ASSOCIATION OF PHYSICAL MEDICINE (BRITISH MEDICAL ASSOCIATION).

THE annual meeting of the Australian Association of Physical Medicine (British Medical Association) was held at Melbourne on September 5, 1946, DR. FRANK MAY, the President, in the chair.

Election of Office-Bearers.

The following office-bearers were elected for the ensuing twelve months.

President: Dr. Frank May.

Vice-President and Honorary Secretary: Dr. B. G. Wade.

Honorary Treasurer: Dr. L. J. A. Parr.

Members of the Committee: Dr. L. T. Wedlick, Dr. A. Lippmann.

President's Address.

Dr. Frank May began his president's address by saying how proud he was to have held the office during the previous year. So much and yet so little had been achieved in the association's first year. So much, in that they now had their own special group, which was a start in the right direction—it was indeed an event to have made a start, for they could on that account look to the future for really big steps forward. So little, because they were, most of them, separated and the year had been spent in organization and their plans were still immature. This term was perhaps wrong, because birth was usually mature; so he would say that they were toddlers. They had to learn to walk and to stand on their own feet.

It was pleasing to think that so many had found the time to come to Melbourne to attend the meeting; it was most gratifying and it was hoped each year to welcome more members. It was a matter for regret that many had not been able to make the journey.

Now that the special group had been started, it should be possible to produce interesting papers at each meeting, and this would help those who wished to learn more of the specialty of physical medicine. Members had to try to keep their ideals. Those present had heard Dr. May speak on the previous night to the Victorian Branch of the British Medical Association on good physical medicine, and it was on this subject that he would again ask for their attention.

As they were medical men, they had all had basic training in anatomy, pathology and physiology, but they should follow up and refresh their knowledge of the anatomy of the skeleton. There were so many cases in which a good knowledge of anatomy was essential. So many patients with joint conditions were brought for treatment and not the least important of the methods of treatment was manipulation.

They were fortunate that they had leaders from whom to learn, and doubly so were they to have Dr. John Mennell as one of that number. They all hoped that he would stay in the "down under" region long enough to give physical therapists the benefit of his experience and perhaps to impart to them some of his illustrious father's knowledge.

Pathology and physiology they had to know so that they could appreciate what was going on inside the body to produce this or that condition which they were asked to treat. Also required was a sound ground work of clinical medicine and surgery, which would come only with experience and reading. They should all beware of becoming too specialized and of not taking an interest in the work of others. An excellent method was to attend staff rounds and staff meetings; it was seldom that one came away from them without learning something of value.

In the specialty of physical medicine, it was necessary to keep abreast of the times with physical and technical principles. They should fellow the proceedings of the British Association of Physical Medicine and particularly those of the American school, where such valuable work would come from the Baruch Committee. The work of the many keen sections of physical medicine of the last-mentioned body could be seen in the *Archives of Physical Medicine*, but the "Year Book of Physical Medicine" should not be neglected, for there extracts from all foreign literature could be read as well. They should not hesitate also to make use of all other methods, for example, the use of drugs and chemicals. Also it was essential, in treating rheumatism, to make a study of dietetics. So much of this was done outside the profession that it was necessary, no, it was their duty, to increase their knowledge in this direction.

There was much in the specialty of orthopædics. He would refer to it as medical orthopædics, and if physical therapists studied it they would find that the production of many painful conditions was due to faulty mechanics and the principles were usually simple. Their use of massage also made it necessary that they should know just what to expect from this part of the work. So, armed with as much knowledge as possible, they might as members of the specialty do justice to it.

Practitioners of physical medicine still had a big uphill fight against prejudice, and it could be overcome only by their being well equipped and refusing to do work which in any way smacked of quackery. There was no need for such work as the specialty was becoming more and more an exact science. They knew what this or that radiation could do and was expected to do. They should also know what this or that physical treatment could not do. So, following these ideals, they would gradually overcome criticism and enter the ranks of accepted specialties.

It was to the young men that they looked in the future. A man could be expert by planning his career for physical medicine. He could spend a year or two at general hospitals and then gain his diploma. Then he could attend some of the excellent post-graduate courses in America and follow up with medical orthopædics and any of the other branches which had been mentioned. Present-day practitioners of physical medicine were the beginners. They hoped and looked forward to the future, for it was full of new concepts and new technicalities. If their colleagues were to have confidence in them, practitioners of physical medicine must be able to deliver the goods. The specialty was so broad that it became more and more difficult, for so many branches of medicine needed them. But changes took place. Those present would remember this or that method of treatment—some were no longer customary. But this was progress and they had to be progressive.

As an example more closely related to their work, they might consider the doctrine of rest. As students, most of them had read or been told to read Hilton's "Rest and Pain". Rest did a lot of good, but it could also do a lot of harm. It did good for cardiac patients, but not for those with a sprained joint. Because an impacted fracture of the wrist or of the surgical neck of the humerus had the fragments firm and fixed, it was recognized that movement could be commenced early. So these patients soon came along for physiotherapy and usually they did very well.

From early physiotherapy to improve function, the patient might go on to occupational therapy or to a rehabilitation class and so back to his job. Physiotherapy must be the right of every injured employee. But the control should be in the hands of the medical practitioner. Particularly should it be in the hands of the specialist of physical medicine, whose experience at the public hospitals gave him that broad outlook so necessary to assess all the aspects of a case. Good physical medicine was possible only with trained physicians and trained physiotherapists.

Regarding physiotherapists, it was a pity that there was no reciprocity with the Chartered Society of Physiotherapy in England and that, even in Australia, there was no uniformity of registration. If the course of training for physiotherapy and the regulations for registration in each State were the same, it would help to improve the status of physical medicine. By this means, the public would not be exploited as they were at present. The public ought to be protected from those who claimed to have special knowledge and skill and who said that they could guarantee a cure. The members of the association should, as a body, take a wide view and organize on a national basis the whole teaching and registration of physiotherapists. This was not only their job as medical men, but the politician should realize the position and aid in the solution of the problem. Insurance companies should see that the practice of good physical medicine was the first step to rehabilitation. This would mean that the worker returned to his job as soon as possible with a lessening of compensation and insurance rates and with a raising of his morale. Something was

being done to help him recover, and that something ought to be good physical medicine, with expert physicians and physiotherapists.

The patient in general practice was demanding physiotherapy and the general practitioner was finding less and less time to give it. He had not the knowledge and training, nor had he the facilities or time to devote to learning it. To cloak his ignorance, he allowed the patient to go where he pleased and even, in some cases, referred him to the unqualified, whose status was thereby raised.

If those who practised it organized physical medicine, and if there were enough practitioners with no shortage of trained physiotherapists, the whole status of physical medicine would be raised with benefit to the public, the insurance companies and the medical profession.

All hospitals ought to have a department of physiotherapy with a specialist in charge, and members of the unit should work as a unit just as the orthopædic and other units did, in giving service to all branches of medicine.

The more private clinics that were available to the public and the general practitioner, the better, for the public would then look to practitioners of physical therapy for specialist service and would not be forced to go to the charlatan.

Many medical men, even specialists, seemed to have an erroneous idea of physical medicine. Criticism was rife and unjust. Massage was "spit and rub"—yes, it was in some circles. Movements were passive. In answer to that Dr. May held that the critics should come in and see for themselves. They obviously knew little of physiotherapy. Why did practitioners of physical medicine have such large gymnasia? Also, how was it possible for any joint to be moved and kept free when the muscles were paralysed or when pain was so excessive that it caused a reflex loss of muscle tone and power? Such criticism as Dr. May had heard was always, in his opinion, evidence of ignorance.

The word "rehabilitation" had come into their work very much of late, owing to the recent war. There was nothing new in this except to those who had not known before. But the war had put it on a properly organized basis, in front of the public and political eye. It was good to see it, for the part of physical medicine in rehabilitation was very important—the most important of all. Films and photographs could be seen showing what rehabilitation was doing at the Austin Works. It was natural for this to occur, for the Austin Works employed thousands of people. Was there any place in Australia comparable? How could any small factory obtain efficient physiotherapy and have all the facilities of rehabilitation? What did the future hold for practitioners of physical medicine?

Dr. May concluded by saying that he closed his term of office with regret, but with pride in the knowledge that he had taken some small part in the establishment of the future of physical medicine in Australasia.

Papers and Demonstrations.

A paper prepared by Dr. John McM. Mennell, of Auckland, was read in his absence by Dr. Frank May. The paper is published on page 161.

A paper prepared by Dr. W. G. Rich, of New Zealand, on the working of the *Social Security Act* in the Dominion was read in his absence. Demonstrations were also held at the Alfred and at Saint Vincent's Hospitals.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on August 14, 1946, at the Children's Hospital, Melbourne, DR. A. P. DERHAM, the Chairman, in the chair. Part of this report appeared in the issue of February 1, 1947.

Splenic Anæmia.

DR. ROBERT SOUTHBY showed a female child, aged three years and two months, who had been admitted to hospital on November 25, 1945, with a history of melena followed by coma. There were no significant findings in the past history or in the family history. Anorexia, coughing, irritability and drowsiness had preceded the melena by a few days. On examination, the child was pale and drowsy. Her temperature was 99·6° F., her pulse rate was 116 per minute and her respiration rate was 24 per minute. A soft apical systolic bruit was audible, but though the abdomen was distended, no mass or viscus could be palpated at that stage. The remainder of the examination revealed findings within normal limits. A provisional diagnosis of melena of unknown origin was made and a blood transfusion of Rh-negative blood of group O (IV) was given immediately. The patient's blood group was found to be A (II). After 270 millilitres of group O blood had been given, a further pint of group A blood was run in. A complete blood examination (after the first 270 millilitres of blood had been given) gave the following information: the hæmoglobin value was 48%, the erythrocytes numbered 2,750,000 per cubic millimetre and the leucocytes numbered 11,600 per cubic millimetre; 68% of the leucocytes were polymorphonuclear cells, 25% were lymphocytes and 7% were monocytes. In the blood film the erythrocytes showed some anisocytosis and poikilocytosis with polychromasia. The spleen was palpable on the day after the child's admission to hospital and thereafter, though it never reached large dimensions and varied in size.

On the morning of November 26 she spat out about six ounces of bright blood. Sternal biopsy was carried out on November 27, and a differential white cell count gave the following information: myelocytes 17%, large lymphocytes 15%, monocytes 2%, small lymphocytes 17%, myeloblasts 8%. The total number of leucocytes was not excessive. Numerous nucleated erythrocytes were present. X-ray examination of the chest on November 29 revealed calcified glands at the right hilum and congestion at the base of the right lung. At this time the temperature became elevated, and there were clinical signs of bronchopneumonia. The bronchopneumonia responded well to sulphamerazine therapy. On December 15 the child was discharged home, clinically well. The stools were normal and the hæmoglobin value was 94%. From that time to July 19, 1946, she was observed at intervals in the out-patient department. She was treated with a full diet, iron and vitamins. The blood was examined at intervals and maintained in a fairly satisfactory condition. Both the Fouchet and the Van den Bergh tests produced a negative response on November 4, 1945.

On the morning of July 19, 1946, she awakened with a discharging nose and fever. She had had a troublesome cough the day before. The stools were black and offensive. She was readmitted to hospital. On examination at this time she was pale and restless. The temperature was 102·4° F. and the pulse was rapid and weak. A soft apical systolic bruit was audible on examination of the heart. The hæmoglobin value was 32% and the erythrocytes numbered 1,590,000 per cubic millimetre. In the smear the erythrocytes showed considerable anisocytosis and some poikilocytosis and polychromasia. An occasional nucleated erythrocyte could be seen, and the leucocytes appeared to be in normal proportion. She was given a transfusion of two pints of compatible blood. The melena persisted till July 25. On occasions the blood passed was bright red in colour. The prothrombin index was 100% (normal) and the Mantoux test produced no reaction. At the time of the meeting the child was awaiting splenectomy.

DR. JOHN COLEBATCH said that he was grateful to Dr. Southby for the opportunity of examining the child recently. It would be agreed that the clinical picture presented—that of splenomegaly and anæmia with a tendency to neutropenia and with repeated gastro-intestinal hæmorrhages—conformed to the description usually given of so-called Banti's syndrome. Current hæmatological textbooks led one to believe that this syndrome was rare in young children. However, in 1935 Smith and Farber from Boston had reported fifteen cases in children, about half of whom were under the age of five years at the time of the first hæmatemesis, and perusal of the in-patient records of the Children's Hospital, Melbourne, showed that in the period from 1941 to 1946 there had been five children diagnosed as suffering from Banti's syndrome, and two of these were under the age of four years when the first hæmatemesis had occurred. Splenectomy, incidentally, had been performed in only one of these five cases; the child was aged ten years and had an enlarged liver, and evidently the physician had advised against operation. The spleen of this patient had been examined by Dr. Reginald Webster, who had found in it the microscopic picture of Hodgkin's disease.

With regard to the treatment of Banti's syndrome, Dr. Colebatch said that he believed that physicians and surgeons generally would agree that the results at present were unsatisfactory, to say the least. A number of surveys had been made of the value of splenectomy; but it required an extensive experience to justify any definite statements on the subject. He drew attention to a significant recent report by Whipple in *Annals of Surgery* for October, 1945, describing in brief the work and the conclusions of the Spleen Clinic at the Columbia Medical Centre, New York. This well-staffed clinic had been established in 1928 for the specific purpose of studying the splenopathies. Some 1,200 cases had been investigated there, and more than 300 splenectomies had been performed. From the detailed study of this large

series of cases, Whipple and his colleagues had drawn some valuable and most interesting conclusions. They had found an increase of pressure in the portal venous system (including the splenic vein) in every patient who presented Banti's syndrome—splenomegaly, anæmia, leucopenia, and usually thrombocytopenia with a tendency to repeated gastro-intestinal hæmorrhage. This increased pressure or "portal hypertension" was considered to be the cause of the various features of the syndrome, and it was found to be due to a mechanical block somewhere in the portal vascular bed. The site of the obstruction determined whether the liver was cirrhotic or normal. In 78% of cases the site of the obstruction had been intrahepatic and due to cirrhosis of the liver; in other words, the cirrhosis was the primary lesion. In 22%, or 27 cases, the obstruction had been extrahepatic. In five of these cases (4% of the total) the splenic vein had been the site of the obstruction, and splenectomy had produced a complete and lasting cure in each of them. But in the remaining 22 cases, in which the obstruction was located in the portal vein, splenectomy had been no more effective than in cirrhotic patients. Splenectomy removed some 30% to 40% of the total portal blood and so produced relief for a time, but only until the portal hypertension had been built up again. When the block occurred in the portal vein, it was usually due to organization of a thrombus; but sometimes it was the result of extension to the portal vein of the obliterative fibrosis that normally occurred in the umbilical vein and the *ductus venosus* at birth. It was of special interest to pædiatricians that Whipple regarded this latter occurrence as a common cause of Banti's syndrome in young children. It seemed, therefore, that if these conclusions were correct, there was little chance that splenectomy would cure a young child such as the one Dr. Southby had presented. The surgeons of the Spleen Clinic in New York had conducted a thorough search for more effective means of treating this syndrome. After discarding omentopexy, ligation of coronary veins *et cetera* as unsatisfactory, Blakemore and Whipple had recently perfected a method for the non-suture anastomosis of the portal vein to the inferior *vena cava* using a vitallium tube. This artificial porto-caval shunt or Eck fistula seemed at the moment to be the most promising form of treatment in cases in which the obstruction was not in the splenic vein; but it was obviously a difficult procedure that was still in the experimental stage.

DR. WARWICK SMITHERS quoted an independent series. He said that in 1927 Donald Hunter had reviewed all cases of splenic anæmia for a ten-year period at the London Hospital. These numbered 23. Dr. Hunter concluded that life was not prolonged by splenectomy, and added to this that there was a 30% mortality rate in the post-operative period. He held that the disease was not one of the spleen, but a disease of the whole portal system. He recommended massive iron and transfusion therapy in these cases.

DR. ALAN PENINGTON said that he had read a series of articles dealing with some aspects of this problem in *The Journal of Pathology and Bacteriology* in 1945. Professor Carew had developed a technique of marsupializing the spleen in animals. He produced experimental hepatic cirrhosis in those cases in which the spleen was not connected to the portal circulation. In all cases splenomegaly and cirrhosis in the spleen developed.

DR. J. W. GRIEVE said that Dr. Leslie Hurley, when he reviewed a large series of similar cases at the Royal Melbourne Hospital, had come to the same conclusion as Dr. Donald Hunter.

Dr. Southby, in reply, thanked the various speakers for their helpful suggestions, and especially Dr. Colebatch for his full report. He had suggested that heparin be used to minimize the chances of thrombosis at the splenectomy site.

(To be continued.)

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 15, of January 23, 1947.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The appointments of the following officers are terminated on demobilization with effect from the dates indicated: Temporary Squadron Leader L. L. Edwards (262615), 12th December, 1946, (Flight Lieutenants) G. C. Morrison (257664), 2nd December, 1946, W. R. Pitney (257530), 6th December, 1946, L. L. Lewis (267760), 10th December, 1946, C. D. Swaine (287405), 13th December, 1946.

Reserve: Medical Branch.

The following ex-officers are appointed to commissions with ranks as shown with effect from the dates indicated: (Temporary Squadron Leader) John Thomson Gunther (272756), 1st January, 1947, (Flight Lieutenant) Cyril David Swaine (287405), 14th December, 1946, Brian Walle Costello (255909), 1st January, 1947.—(Ex. Min. No. 6—Approved 22nd January, 1947.)

Correspondence.

CLINICAL MEDICINE AND SCIENCE.

SIR: What is wrong with clinical medicine and where are scientists leading us?

Some forty odd years ago in the Gulf country of North Queensland I diagnosed and treated sufferers from malaria without the aid of a microscope, but with reasonable success. My then greatest diagnostic difficulty was typhoid fever.

In Australia are many ex-service personnel suffering from malaria, the clinical diagnosis of which, on history, signs and symptoms, is reasonably certain, but in the absence of a positive laboratory confirmation, which in itself in no way contradicts the diagnosis, the patient is refused correct anti-malarial treatment. This is a very serious matter, and the sufferers and the medical profession should be enlightened as to the reason for this unfortunate attitude of negation.

This is only one instance of the inferiority of present-day clinical medicine. In a recent report in THE MEDICAL JOURNAL OF AUSTRALIA reference is made to lipiodol X ray of the maxillary antra as being the sole indication as to the necessity for operation. Here again the scientific X-ray findings are of minor importance as compared with careful clinical examination of the patient, history, signs, symptoms and general health.

Yours, etc.,

Ballow Chambers,
Wickham Terrace,
Brisbane.
Jancary 20, 1947.

ERNEST CULPIN.

A POSSIBLE INCREASED INCIDENCE OF SOME DISEASES OF CHILDREN.

SIR: On returning to pædiatrics a year ago, after an absence of six years, I have noticed an apparent change in the incidence of certain diseases. Outstanding among these is the increased prevalence, in Melbourne at least, of meningitis due to the *Hæmophilus influenzæ*.

Another is the increased proportion of illnesses, broadly classifiable as rheumatic fever, but which, in their onset at least, more closely resemble scarlet fever or other forms of toxic erythema of streptococcal origin. The latter observation is purely empirical, not supported by statistics, and is put forward merely to invite comment.

A further observation, and the only one which would justify this communication, is that I have noticed an apparent increase in the incidence of knock-knee (*genu valgum*) in children under the age of four years. This impression is based chiefly on routine examination of children at the Children's Health Bureau, Anzac House, Melbourne, a preventive medicine clinic for children of returned soldiers, conducted by the Victorian Branch of the Returned Sailors, Soldiers and Airmen's Imperial League of Australia since 1932. During this period approximately 8,500 children, mostly between the ages of two and fourteen years, have been carefully examined and records kept. My impression that the incidence of knock-knee has increased is supported by the examination of a few hundred records only. I doubt whether an exhaustive analysis of thousands of records would be of sufficient statistical value to justify the effort, but I am open to correction on this point. Another reason why I have refrained from trying to prove my observation by an extensive analysis of records is that my impression is shared by a number of careful observers in Melbourne and Sydney at least, including several orthopædic experts.

Assuming that our empirical observation is correct, and the increased incidence a reality, then the cause is still uncertain. Several theories have been advanced. Among these are: (i) the increasing practice during the war years of young children standing with their mothers in shopping queues; (ii) the increasing use of concrete and other hard surfaces in paths *et cetera*; (iii) the habit of children in kindergartens and elsewhere of sitting on the ground with their legs everted at the knee joints (that is, the reverse of the oriental squatting position); (iv) a possible deterioration of diet during the war years, leading to an increased tendency to mild rickets (I know of no evidence to support this); (v) the probable increased use of children's "playgrounds" (collapsible wooden enclosures; these tempt the young toddler to pull himself up and to stand or walk round for hours at a time); (vi) the non-availability of children's "pushers" or "go-carts", which may have led to longer walks for the average toddler. Causes (i), (v) and (vi) could all operate through premature and excessive weight-bearing. The knock-knees are secondary to weak and everted ankles in most cases. The remedy is obvious. Cause (ii), if significant, would be difficult to counter. Causes (iii) and (iv) are of doubtful significance, and, again, the remedy is obvious.

The question is interesting rather than important. Experience has proved that a considerable proportion of toddlers with knock-knee deformity tend to become normal without special treatment. Those who do not can be cured by simple orthopædic measures. It is quite certain, however, that not all recover spontaneously, and it is probably wise to commence treatment early in severe cases, and in milder cases which show no tendency to spontaneous improvement.

Yours, etc.,

A. P. DERHAM.

85, Spring Street,
Melbourne,
January 19, 1947.

CERVICAL AND THORACIC INTERVERTEBRAL DISK DISEASE.

SIR: I reply to Dr. Kelly's letter (THE MEDICAL JOURNAL OF AUSTRALIA, January 18, 1947, page 92) for one reason only. I cannot ignore his request for certain proofs on page 92 when I have asked Dr. McKellar Hall for other proofs on page 93.

Complete scientific proof is not possible as medicine is not an exact science. I will state the evidence upon which my opinions are based.

My dermatome chart was based upon some forty surgically proved cases of cervical disk disease (seven personal and the remainder from reliable sources in the literature), upon a further eighty personal cases of patients suffering from root pains in the upper limb and in whom there was radiographic evidence of the existence of a disk lesion, upon two cases of carcinoma of the apex of the lung involving the first thoracic nerve root, upon four cases of *herpes zoster* affecting the cervical segments and upon one case of scleroderma affecting a cervical segment.

My other opinions were based upon 108 surgically proved cases of disk disease, each personally observed by at least three medical men, and upon the radiological investigation of over one thousand patients during the last three years.

May I take this opportunity of stating that I have never discounted the work of Sherrington and Head? At a meeting of the Western Australian Branch of the British Medical Association I commented upon the accuracy of their findings and upon how these findings had been distorted by others. References to my remarks have been published in this journal.

Yours, etc.,

JAMES H. YOUNG.

131, Scarborough Beach Road,
Mount Hawthorn,
Western Australia.
January 25, 1947.

RECURRENT MALARIA.

SIR: On the subject of recurrence of malaria, the following personal experience may be of interest.

After returning from the islands in November, 1945, I took daily "Atebrin" for three months, and subsequently for six-week periods following malarial attacks to July, 1946, when, following a relapse, I was supplied with "Paludrine" by the Repatriation Department.

I took "Paludrine" as instructed for four months, and felt well, put on weight, and had no signs of malaria. In December, 1946, I took the drug until December 10. It was a particularly busy month, and after that date I completely forgot about malaria and "Paludrine". On January 6, 1947, after several days and nights that were exceptionally busy, I had a classical malarial rigor. Blood film revealed benign tertian parasites. Rigors recurred the next night and again on the fifth night, in spite of quinine and "Atebrin". These were quite severe rigors, with temperatures up to 105° F., splenic enlargement, severe vomiting *et cetera*.

That "Paludrine" is effective in controlling malaria is beyond question, but this shows that the period of just over four months is not sufficient to eradicate the parasite. Of course, since the recommended period for suppression with "Paludrine" is six months, I have only myself to blame for this attack.

Yours, etc.,

D. L. GRAHAM.

Tumut Street,
Adelong,
New South Wales.
January 25, 1947.

Post-Graduate Work.

MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

Programme for March, 1947.

Courses for Higher Degrees and Diplomas.

UNIVERSITY classes for higher degrees and diplomas, suitable for Part I of the M.D., M.S., D.G.O., D.D.R. and D.A., and for Part II of the M.S. and D.D.R., will commence on March 10 and continue on Monday, Wednesday and Thursday afternoons till about August. Enrolments should be made before March 1.

M.D. Part II and M.R.A.C.P.

A series of clinical demonstrations for candidates intending to present themselves for the M.D. Part II and M.R.A.C.P. examinations will commence on Thursday, March 6, at 2 p.m., and continue weekly till June 26. It is proposed to limit this class to 26. The fee for the course is £8 8s., and the March demonstrations at the Royal Melbourne Hospital are as follows: March 6, Professor R. D. Wright, "The Application of Physiological Principles to Clinical Medicine: *Dyspnœa*"; March 13, Professor R. D. Wright, "The Application of Physiological Principles to Clinical Medicine: Cyanosis"; March 20, Dr. C. H. Fitts, "Diagnosis of Cardiac Pain"; March 27, Dr. C. H. Fitts, "Principles of Prognosis in Heart Disease".

Course in Radiation in Dermatology.

A series of lectures designed to meet the needs of the younger dermatological specialists will be given at the University of Melbourne at 4.30 p.m. and 7.30 p.m. from Monday to Friday for two weeks from March 2 to 15. Clinical lectures will be given by Dr. R. Kaye Scott. Dr. C. E. Eddy will lecture in physics, and Dr. Ian Heinz will conduct practical demonstrations. The fee for the course is £8 8s.

Late Afternoon Course in Orthopædics.

On March 6 and 13 at 4.30 p.m. in the Medical Society Hall Dr. Bryan Keon-Cohen will continue the lectures on "The Hazards of Athletics and Common Orthopædic Problems", which will commence on February 20.

Continuous Refresher Course.

Clinical demonstrations will continue in March on four days a week, surgery in the morning, medicine in the afternoon. The fee for this course is two and a half guineas a week, and it is now open to all members of the British Medical Association.

Applications for Enrolment.

Applications for any of these courses should be made to the Secretary of the Committee, College of Surgeons, Spring Street, C.1 (JM 1547-1548), at least two weeks before the commencement of the course. The fee should accompany the application, except in those cases in which it is covered by the Commonwealth Reconstruction Training Scheme entitlements, when attention should be drawn to this fact.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Saunders, Humphry St. John, M.B., B.S., 1946 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Selby, George, M.B., B.S., 1946 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Seymour, John Henry, M.B., B.S., 1946 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Shaw, William Robert Morgan, M.B., B.S., 1946 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Siedlecky, Josephine Ursula, M.B., B.S., 1946 (Univ. Sydney), Rachel Forster Hospital for Women and Children, Redfern.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939, of Queensland, as duly qualified medical practitioners:

Carter, Lindsay Vaughan, M.B., B.S., 1942 (Univ. Sydney), 100, Greenwich Road, Greenwich, New South Wales.

Isles, Jack Dennis, M.B., B.S., 1939 (Univ. Melbourne), 616, Lutwyche Road, Wooloowin, Brisbane.

Gillogley, Jack Francis, M.B., B.S., 1941 (Univ. Sydney), General Hospital, Rockhampton.

Allingham, Johnston Morton, M.B., B.S., 1941 (Univ. Sydney), Fletcher Vale, Charters Towers.

Obituary.

ROY LINDSAY PARK.

WE regret to announce the death of Dr. Roy Lindsay Park, which occurred on January 23, 1947, at Melbourne.

DAVID JOSEPH CROSSIN.

WE regret to announce the death of Dr. David Joseph Crossin, which occurred on January 27, 1947, at Mudgee, New South Wales.

Medical Appointments.

Dr. E. Hertz has been appointed medical officer of health to the West Kimberley Road Board, Western Australia.

Dr. J. H. Priestley has been appointed Government Medical Officer at Canowindra, New South Wales.

Dr. W. D. K. Craig has been appointed a member of the New South Wales Medical Board.

Dr. R. A. J. Stanton has been appointed Deputy Quarantine Officer at Fremantle, Western Australia, under the provisions of the *Quarantine Act*, 1908-1924.

Dr. Z. Fischer has been appointed Quarantine Officer at Onslow, Western Australia, pursuant to the provisions of the *Quarantine Act*, 1908-1924.

The following have been appointed honorary clinical assistants at the Royal Adelaide Hospital: *Neurosurgical Section*, Dr. E. D. Boer; *Gynæcological Section*, Dr. A. D. Byrne, Dr. H. F. Hustler, Dr. H. E. Pellew, Dr. R. L. Verco.

Books Received.

"The 1946 Year Book of Radiology, July, 1945-June, 1946"; Diagnosis, edited by Charles A. Waters, M.D., Associate Editor, Whitmer B. Firor, M.D.; Therapeutics, edited by Ira I. Kaplan, M.D., Associate Editor, Sidney Rubenfeld, M.D.; 1946. Chicago: The Year Book Publishers, Inc. 9¼" x 6¼", pp. 464, with many illustrations. Price: 28s.

"A Textbook of Clinical Neurology", by J. M. Nielson, B.S., M.D., F.A.C.P.; Second Edition, Revised; 1946. New York, London: Paul B. Hoeber, Inc. 10" x 7", pp. 712, with 190 illustrations. Price: $7.50.

"Chemotherapy Yesterday, To-day, and To-morrow", the Linacre Lecture delivered at Cambridge on May 6, 1946, by Sir Alexander Fleming, M.B., F.R.C.S., F.R.C.P., F.R.S.; 1946. Cambridge: At the University Press. 7½" x 5", pp. 40, with illustrations. Price: 2s. net.

"A Synopsis of Surgical Anatomy", by Alexander Lee McGregor, M.Ch. (Edinburgh), F.R.C.S. (England), with a foreword by Sir Harold J. Stiles, K.B.E., F.R.C.S. (Edinburgh); Sixth Edition; 1946. Bristol: John Wright & Sons Ltd. London: Simpkin Marshall (1941) Ltd. 7½" x 5", pp. 730, with 699 illustrations by Dr. E. A. Thomas. Price: 25s. net.

Diary for the Month.

FEB. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 14.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 17.—Victorian Branch, B.M.A.: Finance Meeting.
FEB. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 20.—Victorian Branch, B.M.A.: Executive Meeting.
FEB. 25.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 26.—Victorian Branch, B.M.A.: Council Meeting.
FEB. 27.—South Australian Branch, B.M.A.: Clinical Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

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